IN THE DISTRICT COURT OF THE UNITED STATES
DISTRICT OF SOUTH CAROLINA
CHARLESTON DIVISION

HASKINS, et al.,	)	2:15-cv-02086-DCN
	)	3:15-cv-2123
Plaintiffs,	)	
	)	Charleston,
VS	)	South Carolina
	)	February 28, 2017
3M COMPANY, et al.,	)	
	)	
Defendants.	)	

TRANSCRIPT OF HEARING
BEFORE THE HONORABLE DAVID C. NORTON,
UNITED STATES DISTRICT JUDGE

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Proceedings recorded by mechanical shorthand, transcript produced by computer-aided transcription

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THE COURT: Okay. I'm ready when y'all are.

MR. SWETT: Good morning, Your Honor.

THE COURT: Good morning.

MR. SWETT: Keith Swett on behalf of Haskins. If it pleases the Court, I think we've agreed to do one proceeding direct, and Haskins direct and Chesire and cross and cross, and redirect.

THE COURT: Okay. That's fine.

MR. SWETT: I have with me Nate Finch from Motley Rice, and John George from Waters & Kraus.

THE COURT: Good morning.

MR. FINCH: I have been admitted in Haskins. I would like to enter an appearance on the Chesire case, and Mr. George would like to enter an appearance in the Haskins case so that we can streamline this and make it more efficient with the Court.

THE COURT: That's fine with me. I guess you have to give us another check to do that.

MR. FINCH: We'll be happy to do it.

THE COURT: We'll be happy to take your check. You get pro hac, you make the check and it clears, you are in. I don't think we do, but I'll let you in for the purposes of this hearing anyway. Good. Thank you.

MR. FINCH: Your Honor, at this time the plaintiffs would call Dr. Carlos Bedrossian.

THE COURT: Okay. Dr. Bedrossian.

MR. FINCH: May I stand at the lectern?

THE COURT: You can stand anywhere you want to.

THE CLERK: Please come here and be sworn. Place your left hand on the Bible, and raise your right hand.

THEREUPON:

CARLOS W. BEDROSSIAN, M.D.,

called in these proceedings and being first duly sworn testifies as follows:

THE CLERK: You may have a seat.

THE WITNESS: Good morning, Your Honor.

THE COURT: Good morning. How are you?

MR. FINCH: May I approach?

THE COURT: Sure.

Thank you. More binders.

MR. FINCH: May it please the Court? What we have done, since this is a hearing pursuant to Federal Rule of Evidence 104 about the qualifications and reliability of a witness, we have -- Dr. Bedrossian is prepared to testify. He has assisted us in the preparation of a PowerPoint. Also in the materials that we have just handed up to Your Honor is Dr. Bedrossian's curriculum vitae, his report in the Haskins case, his report in the Chesire case, as well as much of the literature that is discussed in the PowerPoint. Perhaps not all of it but a good chunk of it, which is either cited in

his report or cited in the Dail and Hammar pathology textbook, which he relies on, cited in the materials that we submitted to the Court.

So what I would like to do is conduct the direct examination, establish the qualifications of the witness, go through some general topics, and focus in on Mr. Haskins, and Mr. George will follow up on questions of Mr. Cheshire.

THE COURT: When we get done, can you download a copy of the PowerPoint to a thumb drive, so we can have a copy?

MR. FINCH: Absolutely.

THE COURT: That's fine.

MR. FINCH: We'll give you an electronic copy. The best laid plans of mice and men, we had a PowerPoint, but the computer technology, it was my fault, didn't work. So I'm going to old school it and lay stuff on the Elmo.

DIRECT EXAMINATION

BY MR. FINCH:

Q. Dr. Bedrossian, can you see the screen in front of you?

A. Oh, yeah, I do.

Q. All right. Dr. Bedrossian, could you introduce yourself to the Court and tell us where you grew up.

A. Okay. My name is Dr. Carlos W. Bedroussian. I'm a pathologist, and I practice in Oak Park, a suburb of Chicago.

Q. Where -- could you give us a brief rundown of your

education and tell us where you got your medical degree.

A. After a biological science degree, I attended the University of Sao Paulo in Brazil for six years, and I graduated as a medical doctor in 1967. In 1969, I came to the United States. I served a pathology residency at Baptist Memorial Hospital in Jacksonville, Florida, which now is a Mayo Clinic institution. I also did two fellowships, one in oncologic pathology, meaning study of cancer disease at M.D. Anderson Cancer Center in Houston, followed by a fellowship in pulmonary pathology and cytopathology at Baylor College of Medicine.

I have been certified in anatomical pathology, clinical pathology, cytopathology by the American Board of Pathology, and by the International Academy of Cytology.

Q. Who was Dr. Greenberg, one of the doctors that trained you?

A. Dr. Greenberg was the pulmonary pathologist at Baylor College of medicine. During my training, he had just started a longitudinal study of the Tyler plant in Texas, which was to follow patients exposed to asbestos from, you know, all the way from early in the process; in other words, asymptomatic patients, later on sputum cytology to see if they had asbestos bodies. Later on pathological examination of the lungs when they die and were at autopsy, or when there was a surgical procedure.

Q. And were you involved -- did that series of studies result in papers that were published in the literature?

A. Yes, they did. But, you know, like I was there, and I was leaving as the project was starting. So subsequently, I didn't harvest the seeds that I planted because somebody else was there.

Q. In addition to your medical degree, do you have any other postgraduate degrees?

A. Yes. I received a Ph.D. honoris causa from the University of Naples in Italy based on my contributions to the field of environmental pathology. And I did nonmedical degrees linked to my military service. I retired a a colonel in the U.S. Air Force Reserve, and during my period of serving, I got a college graduation, and I also pursued two master's degrees, one in military studies and the other one on air power.

Q. Are you familiar with something called "The Armed Forces Institute of Pathology"?

A. Yes, I am.

Q. Did you have some role with respect to that?

A. Yes. Like when you are a reservist, you can do your training in different facilities. Although my days was in Belleville, Illinois, I did my annual training at the Armed Forces Institute of Pathology several times, and then at the end of my career, I served six months attached to the AFIP

where my interests was to study pulmonary diseases, including asbestos-caused diseases.

Q. Including mesothelioma?

A. Yes. I saw upwards of 400 cases when I was there.

Q. You reviewed over 400 cases of mesothelioma when you were at the Armed Forces Institute of Pathology?

A. Correct. Extended over the annual trainings for several years, and then the longer period at the end of my assignment.

Q. Could you give the Judge just a brief rundown of the specific education and training you've had with regard specifically to asbestos-related diseases. You've touched upon some of it, but just in a little bit broader overview, could you explain that?

A. Well, in the process of acquiring knowledge about asbestos-related lung disease, I pursued education, training, acquired knowledge and experience, and my knowledge was acquired from my -- you know, following my education and training by practicing pathology and with an interest in environmental pathology, including asbestos-related lung disease. During that process, I had fellows, these are residents that came in for specialty training, and over my career I graduated 36 fellows. Then I set up programs in cytopathology for the diagnosis of mesothelioma by noninvasive procedures. And for 30 years, from 1976 to 19 --

20 years -- 1996, I was on the faculty of the American Society of Clinical Pathology, where I gave workshops on the theme of pulmonary pathology including mesothelioma. And during these sections, I interacted with practicing pathologists who participated in the workshop.

Q. Have you ever served on the faculty at any medical schools or medical colleges?

A. Yes, I did.

Q. Could you describe briefly what that has been.

A. My first appointment was University of Oklahoma. Then I went back to Houston, and I didn't go back to Baylor, I went to the University of Texas at Houston. Subsequently, I was on the faculty at Vanderbilt University, St. Louis University, Northwestern University.

Q. Have you been licensed to practice medicine in various states?

A. Yes, I am.

Q. And where are you licensed to practice and where have you been licensed to practice?

A. I'm licensed in Texas and in Illinois.

Q. While you were at the Armed Forces Institute of Pathology, how many cases of mesothelioma did you review?

A. All together upwards of 400 cases.

Q. And have you consulted on mesothelioma cases since then?

A. Yes, I have.

Q. Approximately how many cases of mesothelioma have you seen while you have been in private practice working on legal cases like this?

A. Approximately another 400 cases.

Q. Have you published any articles in the peer-reviewed medical literature?

A. Yes. I have published a total of 220 articles.

Q. Of those 220 --

A. Book chapters.

Q. 220 articles and book chapters?

A. Right.

Q. How many articles deal specifically with asbestos-related disease?

A. From the diagnostic point of view, in other words, how do you recognize mesothelioma by cytology or histopathology, probably 40 or more papers, and 20 of those were more specific on mesothelioma. And I must add that on the basis of my studies and contributions, I'm a fellow of the American College of Chest Physicians, which is designed for clinicians, but some pathologists who contribute to the field receive that title.

Q. The American College of Chest Physicians, is that related to the American Thoracic Society?

A. No, it's a different one.

Q. Do you -- do you regularly review the literature on

asbestos and asbestos disease causation?

A. Yes, I do. I follow it as a user of the information, but for 32 years, I served as the editor in chief of *Diagnostic Cytopathology.* That is a journal that I founded in 1985, and in that capacity I reviewed articles that are submitted for publication, judging the quality, the accuracy, and the integrity of the information.

Q. Do you maintain a library in your office of textbooks and medical journals and other types of articles that relate to the diagnosis and causation of asbestos-related diseases?

A. Yes, I do.

Q. Do you -- as part of your training and part of your review of the literature, do you have an understanding of the amount of asbestos that is necessary to cause different diseases, including mesothelioma?

A. Yes.

Q. Have you kept up with the literature concerning fiber release from different types of asbestos-containing products?

A. Yes, I did.

Q. Are you specifically familiar with the industrial hygiene and medical literature relating to gaskets?

A. Yes.

Q. Do you have, in fact, a bibliography of gasket literature that you brought with you to your deposition in either the Cheshire case or the Haskins case?

A. Correct. But I should add that not being a materials scientist or an industrial hygienist, I am not an expert on the techniques in the calculations that they use for evaluating exposures.

Q. But do you rely on the information that is generated from industrial hygiene reports or material scientists' reports to obtain a qualitative understanding of the types of exposures associated with working with those products?

A. Yes, correct. So they help me to form my understanding of the connection between the possible cause and the disease.

Q. Have you helped me and Mr. George prepare a slide show that lists the types of materials that you and other doctors rely upon in assessing causation of mesothelioma?

A. Yes, I did.

Q. Is this a list of the kinds of materials that you consider in assessing causation in a mesothelioma case?

A. Correct.

MR. FINCH: Your Honor, I could have Dr. Bedrossian identify and talk about these, but in the interest of time, unless Your Honor has questions about what these materials are on, I could move on.

THE COURT: That's fine. You can move on.

MR. FINCH: Okay. Thank you, Your Honor.

THE COURT: If the defendants think it's important, they can bring it out.

BY MR. FINCH:

Q. Dr. Bedrossian, are you familiar with this textbook?

A. Yes, I am.

Q. *Dail and Hammar's Pulmonary Pathology: Neoplastic Lung Disease*?

A. Correct.

Q. Do you rely on it as reliable on the diagnosis and causation of mesothelioma?

A. Yes, I do. In fact, I contributed a chapter to the first edition of that book.

Q. And am I correct that the chapter on -- the chapter you would find is a very long, lengthy discussion of mesothelioma is Chapter 43, "Neoplasms of the Pleura"?

A. Correct.

Q. That begins on 558?

A. As far as I can see from here.

Q. And it goes on, well, over the footnotes and the citations, ends at page 734 --

A. Right.

Q. -- in that chapter? And does this chapter contain information on all of these subjects: medical literature, industrial hygiene literature, material science literature, discussion of scientific consensus on various things related to asbestos and mesothelioma?

A. Yes, it does. That means a holistic approach to the

study of a disease, not only from the clinical and pathological aspect, but also on the epidemiological aspect, consensus reached by learned bodies that evaluate the available information. It cites case reports and case series which are clinical reports of real patients. It relies on review articles, many of which are meta analysis. It takes into account scientific organizations and regulatory agencies that issue a statement or a consensus paper on that. It takes into account toxicology as well as animal studies, and it touches on industrial hygiene and material science; and more importantly, it addresses the issue of fiber release and the tests of workplace by researchers or governmental agencies.

Q. And is -- is this a pathology textbook that is written by pathologists for pathologists to use and rely upon?

A. Correct.

Q. And you regard it as reliable and authoritative?

A. Yes, I do, although I don't agree with every statement in the book.

Q. When I ask you if it is reliable and authoritative, not every word written, something that you would regard as a well-recognized article or book on the subject matters we are talking about today?

A. Correct.

Q. And is relying on both the statements in Dale and Hammar

and the information that is provided in it part of the methodology that you use to reach conclusions about causation of mesothelioma?

A. That's correct.

Q. And is it part of the methodology that you used to reach conclusions about the causation of mesothelioma in both the Haskins and the Cheshire cases?

A. Yes, it is.

Q. Okay. You were -- you were hired by my law firm in the Haskins case, and Mr. George's law firm in the Cheshire case; is that right?

A. Correct.

Q. And just generally, briefly, what were you asked to do in those two cases?

A. I was asked to confirm the diagnosis and to determine what caused the condition, in this case mesothelioma.

Q. Now, are you familiar with the phrase in medicine generally -- we'll get to asbestos specifically in a little bit -- but "differential diagnosis"?

A. Yes, I am.

Q. Could you explain to His Honor what does differential diagnosis mean both in the terms of choosing between two different diagnoses, and in, once you have the diagnoses, determining what caused a particular disease. Let's start out general and then we'll move specific to asbestos.

A. Yes. That name means that when you examine a case or a situation, you take into account other possibilities, so that you can differentiate between one and the other one before you accept the diagnosis. And that is done through normal clinical practices. For example, you always obtain a history of the disease as the patient remembers. You do a physical examination or you complement that physical examination with ancillary tests, including x-rays, CT scans, MRI, and this is known as clinical pathological correlation.

In the case of environmental lung diseases, you take into account the latency period; that is, the time that lapses between first exposure to a particular agent that is purely the cause of the disease and the diagnosis of the disease. In addition to that, you end up doing an environmental differential diagnosis, where in your mind you consider every possible cause, may they be physical, chemical, or biological; biological being an infection, chemical would be the effect of the substance upon the cellular structure of the lung, and can be radiation, can be asbestosis, can be a number of minerals that affect the lung.

After you excluded all of the possibilities and you are left with asbestos, for example, then you can make your final diagnosis through that entire process from multidisciplines and a holistic approach.

Q. And in a differential diagnosis process, and I'm using

the term to include not only choosing between two diagnoses, but determining causation, is that something that is generally accepted in the medical profession as a way to both investigate what disease a patient has and what may have caused his or her disease?

A. Yes. The first exercise, meaning reaching a final diagnosis is necessary for you to have the exact condition that affects the patient. Next very obviously, the patient and the clinician want to know what was the cause of that condition. And that is when you have to consider all possible physical, chemical, and biologic agents. And after you exclude several of those, you are left with the condition that through your understanding you connect as a causation.

Q. And did you -- did you apply the differential diagnosis process in both Mr. Haskins' case and Mr. Cheshire's case to both confirm their diagnoses of mesothelioma and to determine what caused their mesotheliomas in each case?

A. Yes, I did.

Q. And that was -- was that one of the bases for your conclusions and opinions in both cases, the following this differential diagnosis process you just described both as what the disease is and the causation?

A. That's correct, and you can refer to that as the methodology that you apply through the situation to reach a conclusion.

Q. Are you familiar with something -- we'll get to it later on, but are you familiar with something called the "Helsinki Criteria" for the diagnosis of asbestos-related disease?

A. Yes, I am.

Q. Is that another bases or methodology that you applied to asbestos exposures in both of these cases to form your views in the causation of these cases?

A. Correct.

MR. FINCH: Your Honor, I don't believe for the purposes of this hearing there is any challenge to the diagnosis of mesothelioma in either case.

MR. MERIWETHER: Your Honor, though I think that there are potential issues with the diagnosis in the Haskins case, they are not relevant to this hearing. I don't believe our motion addressed that.

MR. FINCH: That was --

MR. MERIWETHER: Frankly just about *Daubert*.

MR. FINCH: That is my understanding.

MR. FUSCO: The same for the Cheshire case.

THE COURT: There is no issue about qualifications, either, is there?

MR. FUSCO: In the Cheshire case, we don't have an objection to Dr. Bedrossian's qualifications as a pathologist to diagnose mesothelioma. The objection is to the ultimate conclusion.

THE COURT: Right.

MR. FUSCO: Subsequent opinion.

MR. MERIWETHER: Same is true for Haskins.

MR. FINCH: Okay. Your Honor, at this time I would ask you to recognize Dr. Bedrossian as an expert in pathology, the diagnosis of asbestos-related diseases, the causation of asbestos-related diseases, general causation of mesothelioma, and specific causation of mesothelioma.

THE COURT: Okay. Without objection as to qualifications, I understand your challenges to ultimate opinion. Okay.

MR. FINCH: May I proceed, Your Honor?

THE COURT: Sure.

MR. FINCH: Okay.

BY MR. FINCH:

Q. Just briefly, Dr. Bedrossian, am I correct that for both Mr. Haskins and Mr. Cheshire you reviewed pathology material and confirmed the diagnosis of mesothelioma in both cases?

A. Yes, I did.

Q. And then you determined what caused their mesothelioma in each case; is that right?

A. That's correct.

Q. Okay.

A. But I must add that when I made the diagnosis, it doesn't mean that just I look under the microscope and came up with

an answer. I took into account the clinical, pathological, radiological findings, and all the other items that we have discussed before, meaning the work history and the differential between possible causative agents.

Q. We'll get to that in just a second. First, does this slide as a general matter describe the types of materials that you reviewed for these cases?

A. Yes. And, for example, the medical records will contain the history, the physical findings, the radiological features of the case. The pathological material actually provided me with the very lung tissue of that particular patient where I look for abnormalities. And if I find a growth of tumor cells, obviously I make the diagnosis of malignancy, and then depending on the characteristics of the growth, I make a histopathological diagnosis, let's say of mesothelioma, and then I confirm that by special stains that reinforce the original clinical pathological diagnosis.

Q. Now, as part of your review of medical records and clinical records, did you find that these gentlemen suffered from diseases in addition to mesothelioma that are asbestos-related?

A. Yes. They had some, you know, comorbidities.

Q. Did either one of them or both of them have what is called pleural plaque or pleural thickening?

A. Yes. These are the radiological features I was referring

to, and in particular in mesothelioma and in these two patients, there were signs that the patient was exposed to asbestos. These signs are very specific for asbestos exposure to the point that pleural plaques, which are bona fide evidence of asbestos exposure, are not found in other types of scarring of the lung. So in both gentlemen, I saw evidence of pleural plaques.

Q. Is -- is this an example on the right of -- not in these particular gentlemen. This is an example of the pleural plaque, a picture of it taken from an American Thoracic Society document?

A. Yes. And this is a great example of the correlation that I was talking about. If you look at the x-ray, if you look at the outer surface of the lung, you can see that on the left side, you have real evident plaquing occurring in the superior aspect of the lung.

Q. Here, I mean on the --

A. Right.

Q. -- the pleural plaques are on the pleura, around the lung?

A. The outer part of the lung. Now, the lung parenchyma could have had asbestosis, but it didn't in this particular case, but it's not necessary that you have both asbestosis and pleural plaque. Now, the -- from the radiological picture, now you see if the pathology fits that clinical

diagnosis based on radiology. And you see on the right side is the surface of the lung with the impression of the ribs upon the organ, and you have a whitish elongated spot.

Q. This right here, what my finger is on?

A. Yeah. It looks milky white, and when you do autopsies, and you see it, you immediately recognize it as pleural plaque.

Q. So what the laser pointer is pointing up to here, can you see that?

A. Yes.

Q. That is a pleural plaque?

A. Correct.

Q. And you found that in both of these patients, Mr. Haskins and Mr. Cheshire?

A. Yeah, I found them referred to or documented by radiological means. I did not have the gentlemen's lungs for me to examine.

Q. Could you just briefly give us an overview of what is -- what is asbestos and how it impacts on the human lung and pleura?

A. Yes. Asbestos is the general name that you apply to certain fibrous minerals that occur in nature. You have to mine them to find areas where there are outcroppings of asbestos. You crush the rock, and you look at the particles that derive from that, and they are distinctively elongated

where the length is several times greater than the diameter. That defines a fiber.

Q. Now, as part of your work as a pathologist, have you become familiar with the various types of asbestos that were used commercially in the United States and around the world?

A. Yes, I have.

Q. And you take the different types of asbestos into account when you were assessing causation of asbestos-related diseases in a particular person?

A. That's correct.

Q. Could you just briefly describe for the Judge what are the major asbestos varieties that have been used commercially in this country --

MR. FINCH: May I approach the witness?

THE COURT: Sure.

THE WITNESS: Thank you.

A. There are two major types of asbestos. One is called "serpentine," meaning that is curved, which is represented on the left with the word "chrysotile," and you can see that the shape is circuitous. Then there is another category that's an aggregate and known as amphibole asbestos where the fibers are thicker, and they are straight. The important thing is on the left, the chrysotile, because of the shape, is flexible; whereas the materials that use amphibole, the asbestos fibers are brittle.

Q. And so this is -- this is the -- on the -- to the right here, this would be the amphiboles and this is chrysotile?

A. Correct.

Q. Is tremolite -- what is tremolite, and why is it both on the amphibole side and in the little box of chrysotile?

A. Can you repeat that?

Q. What is tremolite, and how does it relate to chrysotile?

A. Tremolite is another type of amphibole. Amosite and crocidolite were commercially available and used widely. Tremolite, on the other hand, can be present in the mixture of fibers. It can contaminate both chrysotile -- I mean a product that contains chrysotile, or it can be part of the mix where the various fiber types are present.

Q. Do you have, based on your review of Dail and Hammar's pathology textbook and other sources, have an understanding of approximately -- of the types of asbestos that were historically used in the United States, how much was the serpentine and the chrysotile, and how much was the amphiboles?

A. At present 95 percent of the asbestos products available on the market contain chrysotile. In the past amosite and crocidolite were common types of asbestos in the products.

Q. Now, you were mentioning how chrysotile is flexible. And could you describe for us the significance of that when asbestos fibers are inhaled into the lungs and what that

means for the chrysotile getting to the parts of the lungs that can cause disease?

A. Yes. In order for fibers to gain access to the lung, first they must be present in the product at hand. Then they have to be friable, meaning the material's friable and breaks up and releases dust particles. Next that -- the particles have to be airborne for the individual to be exposed to it. Like undisturbed, let's say dust on the floor or dust on the surface, by itself is not toxic because it does not gain access to the lung. However, if they are suspended in the air, as determined by seeing visible dust, the individual working with it cannot help but breathe them in their lung.

Now, what happens on the way between the source of the asbestos and the lung cells are different events. For example, the fibers tumble, so they don't particularly go in a direct route, but they also line up with the long axis of the airway, so that even a long fiber can penetrate because it acts like a javelin and travel vertically. On the way to the lung, they encounter bifurcations, meaning splits in the airways, so they usually have an impact in that area. And the impact can cause them to break.

So you may start with a mixture that has a lot of long fibers, but you may end up with small fragments, and that is important because when chrysotile is fragmented, if you have like a curve like that, a segment of that becomes

now straight. So although it was claimed that chrysotile behaved differently than amphiboles on the way to the lung, now we know that because of the overwhelming number of fibers in all the possible events, possible impactions, and fiber in breaking up, you end up with a mixture of fibers in through the lung parenchyma.

Q. What does that mean for the fibers that ultimately get to the pleura where mesothelioma occurs?

A. Okay. Now, in that picture, if you can point to the lymph nodes.

Q. The lymph nodes are the green dots here?

A. Yeah, like the one on the right is a large one.

Q. That one?

A. Yeah.

Q. Okay.

A. Now, as they go in --

Q. And the "they," you are talking about the fibers?

A. Correct.

Q. Okay.

A. When they go in, they are subject to the structures of the lung. Now, the lung has little pumps that remove material from the airway and sequester them. And this is done by absorption of the fibers into lymphatics. Now, in the lymphatics, some of the fibers are eaten up by cells that attempt to eliminate them, but eventually those cells -- I

mean those fibers that are in the lymph nodes, they travel through the lymphatics to the surface of the lung. Lymphatics are blood vessel-like structures that transport liquid called the lymph in cells.

Q. These green dots are the lymph nodes, and the green lines are part of the lymphatic system; is that right?

A. Correct.

Q. Okay.

A. Now, from there -- after they travel into the pleura, the pleura itself has lymphatics, and the pleura lymphatics pick up the fibers and take them anywhere in the body they enter circulation. So that asbestos is a systemic carcinogen. Those fibers travel to other organs, kidney, pancreas, liver, sometimes eyeball. If the person is pregnant, they travel and affect the fetus.

Q. What happens when the asbestos fibers get to the pleura, though?

A. Okay. That is where the action is. They basically -- if you think of it as a gorilla that is invading the structure, once they get to cell level, that is when they do their dirty work.

Q. And --

A. They are carcinogenic. They trigger a process by which a benign cell that replicates and stops replicating turns into a malignant cell that starts replicating uncontrollably, and

that is the definition of cancer.

Q. And is one of the diseases that asbestos causes the pleural mesothelioma like what Mr. Haskins and Mr. Cheshire had?

A. Correct. The tumors that arise on the surface of the pleura are called "mesothelioma." And the tumors that arise in more central areas of the lung, meaning within the bronchial tree, they are known as bronchiogenic carcinoma, colloquially referred to as "lung cancer."

Q. Okay. And does cigarette smoking have any causative relationship with pleural mesothelioma?

A. Not with pleural mesothelioma, it does have a relationship with lung cancer.

Q. Okay. Now, you found in the medical records evidence that both men had both pleural plaquing and mesothelioma. What was the significance of finding a pleural plaque in both of these gentlemen for you in your differential diagnosis causation analysis? Why was that important to you?

A. Okay. Plaquing is a signal that the individual has inhaled asbestos fibers. It's a very specific condition under the microscope which is associated virtually all of them, with some exposure to asbestos. Now, there are other forms of scars that may resemble a pleural plaque, but for the trained pathologist, the pleural plaque is called active pneumonic, meaning it only occurs with that type of injury.

Same thing with the mesothelioma. The connection between asbestos and mesothelioma is so overwhelming that epidemiologists issue a concept saying, "When you have asbestos exposure and malignant mesothelioma, mesothelioma is caused by asbestos until proven otherwise."

Q. You just described something. Is that called "a signature disease," mesothelioma and asbestos?

A. Yes. It's a signature disease or a signal lesion, like the plaque is a lesion; mesothelioma is a lesion.

Q. And is that something that -- is the fact that mesothelioma is a signalled tumor for asbestos exposure something that you took into account in doing your differential diagnosis for both of these gentlemen?

A. Yes, I did. But I didn't stop there.

Q. What did you do for each case?

A. I did a very careful examination of all the evidence that I have: clinical, radiological, pathological, prior studies showing conditions similar to what the workers experienced, and my general knowledge about epidemiology and about causation.

Q. As part of your process of causation analysis in each case, did you rule out other potential causes of their mesothelioma?

A. Yes, I did.

Q. What are some of the very rare potential causes of

mesothelioma that you ruled out in both of these cases?

A. Okay. I'll get into that. But more importantly, the main point is that once you have mesothelioma, it can either be two causes, either asbestos exposure or no asbestos exposure, meaning idiopathic or spontaneous, meaning that you and I and everybody else in the room could develop mesothelioma even if we are not exposed to asbestos.

Now, as far as other causes that I considered are the ones described in the literature. Mesotheliomas can be caused by nonasbestos conditions, for example, radiation therapy. A woman that has a breast cancer and she gets radiation of the chest and the lung is not properly protected, she can develop lung cancer as well as mesothelioma. Individuals who are injected with a radioactive material through the systemic circulation, the material being known as Thorotrast, they also have developed mesothelioma. I have seen one case of a chronic infection of the lung known as empyema that later on the patient developed mesothelioma without a history of asbestos exposure.

But it's important to say that in a patient that has no history of asbestos exposure, you would consider these other causes. But in an individual that has a bona fide history of exposure, has all the necessary criteria to develop mesothelioma, it will be nonsensical to say that all that asbestos that he inhaled was harmless, and he,

therefore, had a spontaneous mesothelioma.

Q. And in both of these cases, did you write reports where you relayed your understanding at the time of what the general asbestos exposure history was experienced by each patient, Mr. Cheshire and Mr. Haskins?

A. Yes, I did.

Q. Okay. Just a couple of other concepts. Latency. What is the -- what does the term "latency" mean when it comes to asbestos and mesothelioma?

A. It means the time that it takes from first exposure to an agent and the diagnosis of the disease.

Q. I want you to assume that --

A. It can also be an incubation period.

Q. I want you to assume that Mr. Haskins, he was first exposed to asbestos in 1953. Is that consistent -- and he was diagnosed with mesothelioma in 2013. Is that 60-year latency period consistent with mesothelioma in the literature?

A. That's correct. In the literature you see some cases with very small time of exposure, anywhere from days to weeks and months, but the vast majority falls between 30 and 40 years, but the bell curve distribution can have patients with 50, 60, and even 70 years, particularly if they are exposed as a child.

Q. And was Mr. Cheshire's latency period also consistent

with mesothelioma? His first exposure was more than 40 years ago to asbestos. Is that consistent with an asbestos-related mesothelioma?

A. Yes, it fell within the range.

Q. Just briefly, what is individual susceptibility generally, and what does it mean in the context of an asbestos-related disease like mesothelioma?

A. The best way to explain it is, for example, we are all in this room, and a person or more may have the flu. That means that they are expectorating viruses, and we are all exposed to it. But not all of us are going to develop the flu. We may have been vaccinated, or we may be particularly resistant to the development of the flu, meaning that depending on the condition of the patient at the point of exposure, one individual may develop and another one may not.

Q. Does this concept of individual susceptibility apply to cancers like lung cancer and mesothelioma as well?

A. Yes, they do.

Q. For example, I'm sure everyone knows people who smoke lots and lots of cigarettes, two packs a day for 50 years and don't get lung cancer, yet other people might smoke a pack a day for 20 years, and they do get lung cancer. Is that an example of individual susceptibility to lung cancer from cigarette smoke at work?

A. Yes, it is. It's known as the "granddad excuse."

Granddad smoked all his life, and he didn't die of lung cancer; therefore, I can smoke anytime I want.

Q. I can smoke two packs of Marlboros a day, and I'll live forever, right?

A. Correct.

Q. And that same principle applies to asbestos exposure and mesothelioma?

A. Yes, it does.

Q. Are you okay? Do you need to take a short recess or are you --

A. Yeah, I could take a real short one to find my glasses.

THE COURT: All right. Take -- just let me know when you are ready, okay?

THE WITNESS: Great. Thank you.

MR. FINCH: Yes.

(Thereupon, there was a brief recess.)

BY MR. FINCH:

Q. Dr. Bedrossian, before we left off, we were talking about individual susceptibility. I'm going to turn now to a slightly different topic. As part of your differential diagnosis, causation analysis here, did you take into consideration what you know from the literature and from books like Dail and Hammar about what is the nature and level of asbestos exposure that is sufficient to cause mesothelioma?

A. Yes, I did.
Q. And what is the nature and level of asbestos exposure that has been demonstrated sufficient to cause mesothelioma?
A. The level has to do with individual susceptibility, so --
Q. You were beginning to describe the -- what is the threshold for mesothelioma?
A. Well, in general, there is no safe threshold because mesothelioma may have occurred in very small exposures, very low exposures, as little as a few hours or a few days.
Q. Has each of these scientific or regulatory organizations issued statements or pronouncements that has discussed this fact that there is no safe level of exposure to asbestos below which mesothelioma will not occur?
A. That's correct. Now, the regulatory agencies issue different types of statements, but an important one is the OSHA permissible emission level. Many people mistake that to mean that if you are below that level, you are not going to have cancer. They didn't say that. They simply said, "For purposes of regulation, this is what we propose to be the limit," but they never exonerated an exposure lower than the PEL as being safe. So there is no threshold below which you will not develop mesothelioma.
Q. In addition to regulatory agencies like OSHA or the EPA, have there been scientific agencies, regulatory agencies and scientific bodies?

A. Correct. Obviously this is a complex subject, so -- and also contentious, so for that reason groups of scientists from different learned societies or institutes have convened together and have elaborated the guidelines or the current understanding of those, meaning that there is no threshold.

Q. Has the International Agency for Research on Cancer concluded that -- is that an example of a scientific organization?

A. Yeah, that's a scientific organization, and they issued a consensus paper in 2012. Another group is the Helsinki Conference on Asbestos and its Diseases. They actually set out what they call "the Helsinki criteria" where they analyze what evidence you need to connect mesothelioma to the asbestos exposure. There are other bodies or other groups in various European countries.

Q. Is the National Cancer Institute an organization that is a scientific entity here in the United States?

A. Yes, it is.

Q. Have they put out an asbestos fact sheet that states that the overall evidence suggests there is no safe level of asbestos exposure?

A. That's correct. They examined a number of studies, a number of documents, and considered all the information, formed an understanding, and from that they developed a conclusion, and their conclusion is that the overall evidence

suggests that there is no safe level of asbestos exposure.
Q. And do you regard the -- you obviously helped us put together this PowerPoint yesterday, and it cites to a fair amount of literature. Do you regard the literature as reliable and authoritative in terms of mesothelioma causation by asbestos?
A. Are you referring to the references that are quoted?
Q. Yes.
A. They are part of the complete body of knowledge that leads to that conclusion, and they are just examples from -- of the types of papers that are available.
Q. Is the British Thoracic Society an example of a scientific organization in the United Kingdom?
A. Yes, it is.
Q. And have they put out a statement on the level of mesothelioma in the United Kingdom?
A. Yes, they did. They are similar to the American Thoracic Society.
Q. And is this an example of what they have concluded about mesothelioma?
A. That's correct. They concluded that there is no evidence or threshold dose of asbestos below which there is no risk.
Q. How many --
A. I want to go back to the individual susceptibility. That is the main reason why you cannot say a safe dose because you

don't know the makeup of the patient being exposed. You don't know his individual susceptibility.

Q. Have there been -- just big picture, how many articles have been published in the peer-reviewed scientific literature on asbestos and mesothelioma? Are we talking hundreds? Thousands?

A. Oh, thousands if you list everything. The ones that I know are reliable ones would be several thousand.

Q. Are there multiple articles in the medical literature that appear in the peer-reviewed literature that demonstrate that there are people who develop mesothelioma with asbestos exposures as brief as a day to a matter of hours?

A. Yes, there are.

Q. Is this concept that there is no minimum threshold level of exposure discussed in the Dail and Hammar textbook?

A. Yes, it is.

Q. What does Dail and Hammar have to say about the threshold for mesothelioma?

MR. FINCH: This is on page 587 of the Dail and Hammar textbook, Your Honor.

A. Right. They stated that there is no minimum threshold level of exposure to asbestos that has been delineated below which there is no increase in the risk of malignant mesothelioma in most authorities' approach causation of mesothelioma by asbestos from the perspective of a

no-threshold model.

BY MR. FINCH:

Q. Are you familiar with an article published in the peer-reviewed literature almost 50 years ago titled "Mesothelioma and Asbestos Exposure" by the lead authors Jan Lieben and Harry Pistamka?

A. Yes, I am.

Q. April, 1967. Am I correct that that article is a series of mesothelioma cases where they confirm the diagnosis and they describe the exposures that various people have?

A. Correct. That is an example of case series. They describe some of the patients individually.

Q. Could you describe for the Court what the exposure history of patient 4Q in this is?

A. Well, as indicated here, the patient was employed in industrial sales and never had any occupational asbestos exposure, meaning handling material. Extensive questioning revealed that on two occasions several years before, he applied asbestos insulation to boilers in his home, mixing the asbestos cement himself, so he created his own cement by mixing it with asbestos. His total exposure during these applications was only a matter of hours.

Q. Am I correct that asbestos cement, not the insulation, but the cement is typically or almost exclusively made of chrysotile asbestos?

A. Correct. It is.

Q. Are you familiar with an article published in the *British Journal of Industrial Medicine* entitled "Mesothelioma Register 1967/1968" where the lead author is Morris Greenberg?

A. Yes, I am.

Q. This is a different Dr. Greenberg than the doctor that you studied under; is that correct?

A. That's correct.

Q. This was a doctor that had published multiple articles. Does this article have descriptions of both occupational exposures and nonoccupational exposures in leading to mesothelioma?

A. Yes, it does.

Q. And am I correct that in one of the cases, the nature of exposure was sawing up asbestos cement sheets to construct two sheds?

A. Correct.

Q. And what was the duration of that exposure?

A. One day.

Q. And that is treated in the literature as an asbestos mesothelioma?

A. Yes, it is.

Q. Did -- does the United States have an organization called the Department of Health, Education, and Welfare?

A. Yes, it does.
Q. And does that -- did something called NIOSH put out a revised recommended asbestos standard in the mid-1970s?
A. Yes, they did.
Q. Studies of human population carcinogenicity?
A. Yes.
Q. And they list one, two, three, four, five, six -- seven studies -- this is a little bit -- let me see if I can make that bigger.

Am I correct that they said the evidence of association between mesothelioma in past exposures to asbestos, that was the finding in all of these studies? Is that correct, Doctor?
A. Correct.
Q. And in the group of exposure, am I correct they state occupational exposures in some cases as brief as one day, and they have that for each of these seven studies listed in 1976?
A. That's correct.
Q. Is that part of the body of knowledge that you rely on in forming causation conclusions in asbestos exposure of mesothelioma cases?
A. Yes, it is.
Q. In addition to medical journal literature that talks about cases or case series, is there analytical epidemiology

that analyzes from a mathematical epidemiology perspective the nature and level of exposure of asbestos where you have increased risk?

A. Yes. There are case-controlled studies. There are cohort studies. There are population studies, including surveillance programs for countries that have that.

Q. And are you familiar with the -- I believe it's one of the papers you cite is Rodelsperger common 2001?

A. Right. In Germany.

Q. And what was the main conclusion from this German study?

A. They confirmed a very important aspect that I have not talked about yet.

Q. Okay. Explain what they confirmed.

A. Yeah. There is the concept of dose response. Even though all of what we have discussed was supportive of causation, to be absolutely certain that it is the asbestos that is causing the effect, you have to do some calculations showing that when you increase the dose, you increase the response.

Q. On a population basis you are talking about?

A. Population basis, correct.

Q. Explain that.

A. You know, like commonsense will tell you the more of something you are exposed to it, the more effect it will have. For example, alcohol. But in this particular

instance, they correlated the total dose to the increasing effects of that particular agent. In the case of asbestos, that would be asbestosis, or it could be mesothelioma, and a related concept of dose response is that when you have a high dose, the latency period is shorter than when you have a lower dose. So a high dose over a short period of time causes the effect, and lower doses over a longer period of time also cause the effect because the effect depends on the cumulative total dose of the exposure.

Q. And am I correct that the Rodelsperger paper talks about there is a causation even at a cumulative exposure level below one fiber?

A. Correct.

Q. Are you familiar with a paper -- it's in your bibliography -- relating to gasket studies, but a paper entitled "Occupational and non-occupational attributable risk to asbestos exposure for malignant pleural mesothelioma"?

A. Yes.

Q. The lead author is Lacourt in 2014?

A. Correct.

Q. And you regard this as reliable and authoritative on the question of what kinds of exposures can cause mesothelioma?

A. Yes, I do.

Q. And what was the most common type of asbestos used in France during the times they were studying in this paper?

A. Chrysotile.
Q. What does it mean to have an odds ratio of 4.0 for any exposure greater than zero, less than one fiber cc years?
A. Did it say "one and zero"?
Q. No, greater than zero, between zero and .1, there is an odds ratio of 4.0.
A. It means even at the low level, the ratio of the odds that the patient will develop mesothelioma are increased. So even at that low dose, the patient will develop mesothelioma.
Q. Let's talk specifically about chrysotile asbestos, which was the predominant type of asbestos used generally and also asbestos that -- do you understand that chrysotile asbestos was in asbestos-containing gaskets?
A. Yes, I understand that.
Q. Does chrysotile asbestos cause the same asbestos-related diseases as the amphibole asbestos?
A. Yes, they do.
Q. Do chrysotile fibers cause asbestosis?
A. Correct.
Q. Do they cause asbestos-related lung cancer?
A. That's correct.
Q. Do they cause pleural plaques?
A. Correct.
Q. Do they get to the pleura?
A. Yes, they do.

Q. Can they damage chromosomes?
A. Yes, damage cells, including its components, the chromosomes, the DNA sequence, some specific portions of the DNA.
Q. Can chrysotile fibers both be an initiator and a promoter of cancer?
A. Correct.
Q. Explain -- His Honor may not -- maybe I'm assuming too much here, but can you explain to the Judge what is an initiator of cancer? What is a promoter of cancer?
A. Initiator is like a trigger mechanism that provokes a cascade of events that eventually will develop into a cancer. Promoter is that same agent facilitates that progression through the course of the cancer development.
Q. Does chrysotile asbestos act as both an initiator and promoter of cancer?
A. That's correct.
Q. Is chrysotile asbestos genotoxic?
A. Correct.
Q. What does that mean?
A. You know, when the asbestos is presented to the susceptible cells, so let's say when asbestos makes contact with mesothelial cells in the pleura, it will affect -- first create a pathogenetic mechanism whereby inflammation in the oxygen radicals do some harm at the center of that. But it

also affects the cells at the molecular level. It affects the actual genes that are present in a normal cell. What we'll do is we'll kill off tumor suppressor genes that protect the organism from developing cancer and activate genes that become autogenetic and cause the cancer.

Q. And am I correct that it's generally understood that for genotoxic materials, there is no threshold dose below which a cancer will not occur?

A. Correct. It all depends on the agent affecting a cell, making contact with the cell when the cell is in a vulnerable period, the cell cycle; the cells multiply. Now, there is a point of the cell cycle that is susceptible to genetic transformation.

Q. And am I correct that scientific agencies such as the American Thoracic Society have stated that all forms of asbestos can cause both nonmalignant disease and cancers?

A. That's correct. And the explanation is asbestos is both fibrogenic and tumorigenic, meaning it has the capability of creating inflammation and fibrosis, and it has the potential to cause malignant transformation at the genetic level. So virtually all patients will have some inflammatory response to the physical presence of the fiber, but in the susceptive individual, this phenomena triggers the release of certain molecules from the microphage that affect the genetic makeup of the target cell, and that then is the genetic aspect of

the injury.

Q. Are you familiar with the American Thoracic Society's statement on the diagnosis and initial management of nonmalignant diseases related to asbestos?

A. Yes, I am.

Q. This is the consensus document that has the statement that I was just talking about earlier and you were describing earlier about how all forms of asbestos by definition and classification appear to cause malignancy, and all may cause a nonmalignant disease described as well?

A. Correct.

Q. Doctor, have scientific organizations looked at the question of whether chrysotile can cause mesothelioma just by itself?

A. Yes, they have.

Q. And what has been the conclusion of scientific and research agencies about the question of whether chrysotile causes mesothelioma?

A. The basic conclusion would be to say that an asbestos fiber is an asbestos fiber. Some of them may be curved, some of them may be straight, but all of them are poisonous. Chrysotile is less potent a poison, whereas amphibole is a more potent poison. If there is a mixture of the two, both of them contribute to the poisonous final effect, which is what we call the cumulative dose, responsible for the injury.

Q. And am I correct that all of the scientific agencies shown on slide 45, the International Agency for Research on Cancer, the National Toxicology Program, the United States Surgeon General, World Health Organization, the World Trade Organization, the American Cancer Society, all of them have concluded that chrysotile causes mesothelioma and there is no safe level of exposure to chrysotile?

A. That's correct.

Q. Now, have you looked at the question in the literature about whether -- which of the asbestos varieties are preferentially translocated to the pleura?

A. Yes, and that's important because it has to do with the pathogenesis.

Q. Okay.

A. Some individuals believe that the effect of the fiber is related to the fibers that remain in the lung tissue after they are inhaled.

Q. In the fat part of the lung, the parenchyma of the lung?

A. In the surface of the lung. But what is important is whether or not that fiber was present in the material that was inhaled and how much of it was present. The retention of fibers is different. The amphiboles are more retained in the lung. In chrysotile, it has a shorter period of time that they are intact, and then they get removed. So if you go and measure their level after a period of time, obviously there

will be lower concentration of chrysotile, because it already left the lung. So it's the difference between saying, "I'm going to accept this as a poison only if I find residue of the poison in the tissue." It's almost as if because chrysotile is untraceable or partially untraceable, they try to exonerate that fiber from causing any effect.

But there is the concept of hit and run. The fiber as it's passing through the lung and it stayed around for its biopersistency level, it causes the damage, but later on it gets removed. But you don't need to indict that fiber by catching them in the act. You can have a hit-and-run phenomenon.

Q. There is two concepts I want to draw out from you there. One relates to biopersistence in the lung, the other relates to translocation of chrysotile fibers to the pleura. Start with the latter one first. Have researchers at Mount Sinai and elsewhere examined the question of if you actually look in the pleura to find what is there, whether there is more chrysotile in the pleura or other asbestos fibers?

A. Yes, because that is very important. That is where the mesothelial cells are. That is where the target cells are.

Q. Are you familiar with papers by Dr. Suzuki and his colleagues that address this question whether chrysotile is the predominant fiber type found in the pleura?

A. Yes.

Q. Have there been other researchers as well that have found the same?

A. Yes, particularly in France and Italy.

Q. Describe for the Judge what we are talking about here and what the researchers that have published on this have found.

A. If you recall when I was talking about the normal defense mechanisms of the fibers entering and lining up with the longitudinal axis of the airways, so after they get to the central part of the lung, some may stay there; some, however, can travel through the lymphatics.

Q. Is this what we are talking --

A. The center portion of the lung in the pleura. And this process is known as "translocation."

Q. So you were talking about the lymphatic system, these green dots here?

A. Right.

Q. And then this is an example of one of the articles published by Dr. Suzuki and his colleagues. What did they find when they analyzed this question?

A. They found that in dealing with chrysotile that phenomenon took place, and that phenomenon was the major translocation. In other words, chrysotile is more easily translocated to the pleura. And, furthermore, the concentration of chrysotile in the pleura was larger than the amphibole's.

Q. Now, that was a concept of translocation of chrysotile fibers to the pleura. You had also been telling us the concept of biopersistence in the body generally. Do you recall that?

A. Yes, I do.

Q. Okay. Now, does -- I believe -- would you agree that it's generally accepted that cigarette smoke causes lung cancer?

A. That's correct.

Q. Does -- and cigarette smoke biopersistent in the body, is that necessary for it to be biopersistent in the body to cause lung cancer?

A. No, not so, because when the person is smoking, it is the same as if the concentration of carcinogen stays the entire time. Because, you know, a new puff comes in, but the same agents are present. So after they are present, after they made contact with the susceptible cells that became neoplastic, cigarette dissipates. Caught air goes up. So the smoke itself bathed in the lung caused the change, and then it gets, say, now if it is a mixture of particles that are inhaled.

Q. So, say, asbestos fibers of different particles -- chrysotile and amphibole, let's say?

A. Right. If they do the same, if they get inhaled, first they go through the lung. They do their carcinogenic

mechanism, and then they get in the pleura. From the pleura they can go anywhere. They can go into the kidney, liver, thyroid, and you don't need to find the fiber in the lung after the end of the process of that at autopsy to say that that fiber did go through the lung, cause the effect, and is related to the cause.

Q. Did the International Agency for Research on Cancer put out a monograph on asbestos about five years ago that you are familiar with?

A. Yes, they did.

Q. It has --

MR. FINCH: Your Honor, we didn't include the entire monograph because it's hundreds pages thick.

BY MR. FINCH:

Q. Does that monograph address multiple different lines of scientific evidence that relate to the question of asbestos and disease causation?

A. That's correct.

Q. And did IARC again reconfirm in 2012, as they had in the past 40 years, that chrysotile can cause mesothelioma just like the amphiboles?

A. Correct. And presently is the cause of mesothelioma because 95 percent of the asbestos still used is chrysotile.

Q. Has the World Health Organization put out both position statements and monographs on the question of chrysotile

specifically?

A. Yes, they did.

Q. And this is one that was put out in 2006. What did the World Health Organization say in 2006 about whether there was a threshold for chrysotile?

A. Basically they said that occupational exposure to all types of fibers -- they list crocidolite, amosite, and chrysotile -- as well as among the population living in the neighborhood of factories or mines, and people living with asbestos workers, all of those are known to contribute to mesothelioma.

Q. And did they also go on to talk about whether there was any threshold for the carcinogenic risk of chrysotile?

A. Yeah. They reinforced that there is no safe or threshold level below which you don't have mesothelioma.

Q. Are you familiar with the monograph the World Health Organization put out in 2014 dealing specifically with chrysotile asbestos?

A. Yes, I am.

Q. And are these the main takeaways from that monograph in 2014?

A. That is correct, you know, like all of those studies and position papers are -- or reviews address this issue. And essentially one important issue is that chrysotile, despite the fact that it's less potent, is a contributing factor that

can cause mesothelioma. In other words, if you are hit with a 22-caliber bullet or a 30 -- or 38-caliber bullet, both of them can cause death. And there is no level of exposure below which there is no mesothelioma. So there is no safe level of exposure.

Q. Now, as part of your review of the literature, have you collected and put into a binder literature relating specifically to the types of asbestos exposures experienced when people do things like remove gaskets from a flange?

A. Yes, I have.

Q. And have you -- you brought with you to a deposition a list of that literature. I believe you have collected it into a notebook. Is that correct?

A. Correct.

MR. FINCH: Your Honor, I believe one of the two notebooks in Your Honor's materials, we -- as a matter of housekeeping, I'll offer all these as exhibits at the end. There is gasket material literature. I'm not going to take the time to go through all of it.

BY MR. FINCH:

Q. Have you picked out a couple of examples of the fiber levels that you have seen in the literature that you take into account in a qualitative manner in assessing causation?

A. Yes, I did.

Q. Are you familiar with a work practice simulation done by

Millette in 1995 that describes the levels of exposure experienced with doing various things to gaskets, including hand-scraping or sweeping up after?

A. Right.

Q. And these would be asbestos-containing gaskets?

A. Yes.

Q. And does this table on slide 53 have some of the fiber levels that are .14 to -- for the hand-scraping; 5.5 for sweeping of the area after removal?

A. Correct.

Q. And are you familiar with a paper by William Longo and some coauthors where they also examined both the fiber levels associated with using hand scrapers and with using power tools to remove gaskets?

A. Yes. If you will go back to the previous one, they found that power brushing -- I'm sorry -- power wire brushing is higher than hand-scraping, for example, and the method by which you handle the gasket material influences the amount of fiber that are released.

Q. So, Doctor, we are not going to show the Judge the power tool numbers from Dr. Longo's studies, but did he also look at hand-scraping as an exposure in his literature?

A. Correct.

Q. And what was the fiber level per cubic centimeter shown as an example from using hand-scraping both on a short-term

level and a TWA level?
A. Was 10.1 --
Q. And --
A. -- per cubic centimeter.
Q. And do you understand TWA to be time-weighted average exposure?
A. Correct.
Q. And that would mean over an eight-hour day, somebody doing that activity would be exposed to what?
A. 1.5 fiber per cc.
Q. Okay. My son is in the third grade and does a lot of third grade math, but I would like -- having these numbers about fibers per cc, we are talking about -- a cubic centimeter is about the size of a sugar cube; is that right?
A. Right.
Q. Can you -- have you done an analysis to translate that for us into -- if you are in an environment where somebody is breathing asbestos of a level of one fiber per cubic centimeter, what does that mean over the course of one working day?
A. Well, it means that they will accumulate a certain amount of fibers as opposed to somebody else who may be breathing a higher concentration. So the higher the concentration, the more total cumulative dose you accumulate.
Q. Okay. Have you done the math for us that shows how many

fibers someone would breathe into him in the course of an eight-hour day at a one fiber per cc level of exposure?

A. I did that with the help of a calculator.

Q. Okay. Could you explain what we are -- go through the steps here for His Honor and for the Court?

A. Okay. Human beings under normal circumstances or when they are working, when they are active, they take 16 breaths per minute. Each breath brings in a volume of 500 cubic centimeters of air. Now, depending on the concentration of the fiber present in those 16 breaths, you are going to have the fibers per minute that gain access to your lung. So if it is one breath, each one would have one fiber per cc. When you have 16 breaths, the total would be 8,000 fibers per minute.

Now, the individual doing the work stays doing the work anywhere from eight to 12 hours a day so that you are calculating how much exposure occurs during the period of time. For example, here for 15 minutes, they accumulated 120,000 fibers into the lung. And for an hour would be 480, and in an eight-hour workday, there will be eight times the amount per hour, and that would be 3.8 million fibers.

Q. Now, I've heard it said that, particularly in briefs written by defendants in asbestos cases, there -- there -- asbestos is everywhere in the normal air, the ambient air. Do you have -- is there literature that has gone out to

examine in an unpolluted environment, normal ambient air, what the level of concentration of asbestos is in the ambient air?

A. It's very small. So --

Q. And Doctor --

A. The number would be 0.0005. So I don't even know what to call that number.

Q. Point four zeros and a five? This is data from Dr. Roggli?

A. So it would be one four millionth of fibers.

Q. Okay. Dr. Roggli is an expert that often works for defendants in asbestos cases?

A. Yes.

Q. He was actually one of the defense experts in these cases; is that right?

A. Correct.

Q. And were -- who trained Dr. Roggli?

A. Yeah. It's coincidental, but I was the first fellow that Dr. Greenberg had in his program of environment pathology. Later on -- and at that time, Roggli was a medical student. So we crossed paths, but I did not particularly know him at that point. Later on when the study was more entrenched, he was the fellow and did work directly with Dr. Greenberg and went on to publish papers with Dr. Greenberg.

Q. And he's also published a book that has information about

the median asbestos fiber concentration in American cities in an unpolluted environment. That is one of the data sources you considered?

A. He did that in his first book. Dr. Greenberg was a coauthor.

Q. Roggli and Greenberg, *Pathology of Asbestos Disease*?

A. Correct.

Q. Greenberg and Roggli. Have you done the math for us to show if you are not exposed to asbestos occupationally, but instead from ambient, normal, unpolluted air, how many asbestos fibers you would breathe in a 24-hour day?

A. So if you are in resting mode and you are not near any asbestos products, you are not handling it, you are not disturbing it, you would take 12 breath per minute, and using the same calculation, 500 cc of air present, in one minute you are going to have 6,000 cc of air that you inhale. Now, depending on the concentration of the fiber present in the air, in an hour you have 360,000 cc, and because the concentration is 0005 cc -- I mean, 5 per cc, your accumulation would be 8,640, but for in a 24-hour day, you end up with 432 fibers in the lung.

Q. So --

A. You have a considerable lower total dose.

Q. Am I correct that one day of occupational level of exposure, such as scraping gaskets off a flange, you would

breathe approximately 4 million fibers versus 432 if you are not exposed to asbestos?

A. Right, if you determined that the concentration is one fiber per cc. If it's higher than that would be several million.

Q. You -- earlier today you describing the concept of dose response and cumulative exposure when you were talking about the Rodelsperger study. Do you recall that?

A. Yes.

Q. Do you have a graphic for us that shows the dose response curve for mesothelioma?

A. Yes.

Q. Could you explain what we are looking at here.

A. This is showing the greater disease occurring with greater exposure. Greater exposure is in the X axis, so the more exposure you have, the more disease you develop. There is a certain level of asbestos in the air, and that's known as "the environmental exposure," which is very low as we have discussed. If the person is in the home of an asbestos worker who comes back with dust in his clothing, that person would be exposed to a higher cumulative dose than the environment. And that is known as "household" or "take-home exposure."

Likewise, in an occupational setting, obviously you are going to have greater concentration of asbestos fiber.

If you are in the vicinity where that work is being done, but you yourself are not handling the asbestos product, you are a bystander, and you are exposed to asbestos more so than a household person; and more at the top of the scale, someone who is actually doing the work directly in the work space where asbestos is released, that person would have occupation exposure and a greater degree of disease.

Q. Do you need some water, Dr. Bedrossian?

A. I need a lozenge.

Q. We were talking about the significance of increasing exposure to asbestos in terms of a dose response -- are you okay?

A. Yes.

Q. Is there also an effect if you have higher asbestos -- cumulative asbestos exposure, what impact that has on the latency? That means the time period from the first exposure to the development of the disease?

A. The higher the intensity, the less the latency needed. So high intensity over a short period of time can be as great as low dose over a long period of time.

Q. Do you have an opinion in each of these cases as to what caused Mr. Haskins' and Mr. Cheshire's mesothelioma? Do you have an opinion in each of these cases as to what caused Mr. Haskins' and Mr. Cheshire's mesothelioma?

A. Yes, I do.

Q. And what is that -- you were provided information about their asbestos exposure history in both cases, right? Am I correct? We'll get to more detail in that in a minute. What was the cause of Mr. Haskins' mesothelioma?

A. It was the total cumulative dose that they accumulated from various products that they worked on.

Q. How about for Mr. Cheshire?

A. Same thing.

Q. Okay. Is the concept that it's the cumulative exposure to asbestos or cumulative dose that causes the disease discussed in Dail and Hammar's pathology textbook?

A. Yes.

Q. At page 587, what did Dail and Hammar tell us about the implications for when there are multiple asbestos exposures and cumulative exposure?

A. He said that the factor that emerges from the model and its modifications is that when there are multiple asbestos exposures, each contributes to the cumulative exposure, and hence to the risk and causation of mesothelioma within an appropriate latency period. So what he's saying is different exposures are part of this -- the dose exposure. They contribute to the total. It is the sum of all exposures being the total cumulative dose.

Q. In addition to Dail and Hammar, when epidemiology studies measure risk of mesothelioma from asbestos exposure, do they

do so in increments at various cumulative doses?

A. Can you say that again?

Q. In epidemiology studies like Rodelsperger, do they talk about subsets of exposure, or do they talk about cumulative exposure as to what influences the risk?

A. Yes, they do, and that is where the concept of cumulative came from.

Q. Has OSHA, the Occupational Safety and Health Administration, put out a statement on this concept of how every occupational exposure contributes to getting a risk of an asbestos-related disease?

A. Yes, they did.

Q. Would you read for the Court what OSHA had said on its website. I printed this out a couple of months ago. I assume it's still on there today.

A. Well, first they said there is no safe level of asbestos exposure for any type of asbestos fiber, meaning a high-potency one or a low-potency one don't have a safe level below which they don't cause mesothelioma. They also said that asbestos exposures as short in duration as a few days have caused mesothelioma in humans as documented by case reports and case series. They further stated that every occupational exposure to asbestos can cause injury or disease, and every occupational exposure to asbestos contributes to the risk of getting an asbestos-related

disease, meaning it is the cumulative exposure that increases the risk of the patient developing a condition related to asbestos.

Q. Okay. I want to turn to one more topic before we get to your ultimate conclusions in the Haskins case. You were talking about the Helsinki report, and it was published in the peer-reviewed literature. Can you tell the Judge what is the Helsinki conference and what came out of it?

A. This is one example of a scientific consensus conference whereby they bring specialists in different areas, including clinicians, chest surgeons, radiologists, pathologists, as well as experimental researchers who do work in animals and hygienists who do work in an environmental areas. And they get together for two weeks, approximately, and they analyze all aspects of the universe of asbestos exposure in relation to disease.

And, of course, they are following the literature. So they examine the pressing subjects, the topics that are important; for example, which fiber's more potent, what dose is safe or not safe. In other words, they analyze the entire universe of asbestos-related lung disease.

Q. And they published a paper in 1997, and it was republished a few years ago in 2014?

A. That's correct.

Q. And did the Helsinki consensus group conclude that --

like the rest of the scientific organizations -- that all types of malignant mesothelioma can be caused by asbestos with the amphibole showing a greater carcinogenic potence than chrysotile?

A. I would state that all types of asbestos can cause mesothelioma with the amphiboles showing greater carcinogenic potency than chrysotile. Stated the way it is, it's also correct meaning all types of mesothelioma, meaning subtypes like epithelioid, sarcomatoid, or some other type are induced by asbestos.

Q. Did the Helsinki criteria also talk about what is the most practical and useful work of -- useful measure of occupational asbestos exposure?

A. Okay. That is a very important conclusion that they reached.

Q. What is that conclusion?

A. In general, reliable work histories provide the most practical and useful measure of occupational exposure. Now let me explain. Some scientists like to push the concept that in order to determine if there was exposure or not, looking at the lung or quantitative the number of fibers is more important than the presentation of fibers at the enter point, when the person takes the breath.

And I already explained that is incorrect because fibers break, become smaller. They get eliminated. Some of

them are undercounted when you are looking at the microscope. Chrysotile fiber, five micron or less, is not visible by contrast microscopy. Calculations underestimate the number of fibers. And that is the reason why the work history, when taken correctly by someone trained to do an environmental occupational history, is the more reliable or the more representative of the exposure.

Q. And did the Helsinki criteria discuss what should be considered sufficient for mesothelioma to be designated as occupationally related to asbestos exposure?

A. Yes. The duration, what we have been talking about, meaning that an occupational history of brief or low level exposure should be considered sufficient for mesothelioma to be designated as occupationally-related asbestos exposure, exactly for the reason that you have individual susceptibility.

Q. Does this diagram describe the various ways a doctor relying on them can conclude whether or not asbestos exposure causes someone's mesothelioma?

A. Yes. And they can be divided in two types: One, finding the fiber in the lung; and the second, looking at the effects of the fiber. For example, elevate the asbestos burden, yes, this person was exposed to asbestos because I see the asbestos in the lung. Pleural plaques, because it's an effect which is specifically linked to asbestos exposure, is

a signal lesion. It is a landmark that is pathognomonic. When you see it, you can conclude that asbestos caused it. Same thing of asbestosis, meaning a special type of fibrosis or scarring of the lung that has been linked extensively to the exposure to asbestos.

Q. Did -- in the case of Mr. Haskins, first of all, do you need to have all of these in order to attribute a given mesothelioma to asbestos exposure?

A. Correct.

Q. You need to have all of them, or can you have just one of them? Do you need to have all of these things, or is one of them sufficient?

A. No, you don't. But I forgot one item.

Q. Sure.

A. In addition to what I just said, the condition has to have the proper latency period.

Q. The latency from the time of first exposure to the disease has to be consistent with what you said in the literature?

A. Right. In a given case, you may have all of these. In another one, the exposure history is unclear or somewhat not carefully remembered by the patient. But if you find pleural plaque and/or asbestosis, it is a signal that the exposure to -- because many patients don't remember that they were exposed or, you know, because they are unaware that they were

exposed.

Q. Okay. And am I correct that in both Mr. Haskins and Mr. Cheshire, you had information from the medical records that they both had pleural plaques or pleural thickening, and they both had a history of asbestos exposure with a more than ten-year latency period?

A. Yes, I did.

Q. Okay. Now, as part of your report --

MR. FINCH: And, Your Honor, I'm going to ask some specific questions about Mr. Haskins case, and then Mr. George is going to clean up and do some things about Mr. Cheshire.

BY MR. FINCH:

Q. But as part of your work in the Haskins case, did you do a report where you were provided information by my law firm about his -- in general terms his asbestos exposure history? Is that correct?

A. Correct.

Q. And you wrote on pages 1 and 2 of your report in Haskins a summary of that asbestos exposure history?

A. Yes, I did.

Q. Okay.

MR. FINCH: I'm not going to take the time to read the entire two or three paragraphs into the record now. Your Honor will have the report in evidence.

BY MR. FINCH:

Q. Dr. Haskins -- excuse me.

Dr. Bedrossian, in addition to the facts in your report, I want you to assume the following facts about Mr. Haskins' asbestos exposure from removing asbestos gaskets from Buffalo pumps we believe will be shown by the testimony of the fact witnesses at trial. I want you to assume that Mr. Haskins served in the United States Navy from January 1953 to May 1956 and served aboard the USS *Cony*. I want you to further assume that Mr. Haskins was a fireman assigned to the forward engine room. I want you to assume for a three-month period of time, his regular duty on a daily basis was to assist the mechanics in repairing Buffalo pumps in the engine room. I want you to assume that Mr. Haskins' job every day for three months was to use a scraper and a wire brush to remove old asbestos-containing gaskets from Buffalo pumps which took approximately one hour per gasket, and he did this during his regular work day for approximately a three-month period of time.

Finally, I want you to assume that this process created visible dust which Mr. Haskins inhaled. And, finally, I want you to assume that he was also exposed to asbestos from asbestos-containing insulation onboard the ships which was disturbed either when the ship was overhauled and repaired at sea or when the ship was vibrating. Do you

have those facts in your mind?

A. Yes.

Q. Okay. Focusing first on the total of the history, both in your report and what I just read to you, what conclusion do you draw as to what caused Mr. Haskins' mesothelioma?

A. It was the cumulative exposure derived from the activities that you described where he handled asbestos-containing materials and created visible dust that he inhaled.

Q. In reaching that conclusion, did you rely upon the Helsinki criteria and apply the Helsinki criteria to his situation?

A. Yes, I did. As one of the tools, yes.

Q. Did you also apply the differential diagnosis approach that you talked about earlier this morning?

A. Yes, I did.

Q. Okay. I want to focus you on just a separate hypothetical. I want you to assume that you were presented with a patient A who has been diagnosed with mesothelioma, and 50 years before the only asbestos exposure Mr. A had was using tools to remove gaskets from Buffalo pumps flanges for three months, asbestos gaskets. He had the exact same exposure to Buffalo pumps asbestos-containing gaskets, but he is a different patient. He has mesothelioma, and it's a 50-year latency. Based on the Helsinki criteria and your own

differential diagnosis process, would you say that that is an asbestos-related mesothelioma?

A. Yes, it is because there is nothing else to be an alternative cause for that mesothelioma.

Q. Okay. Now, I want you to now focus back in on Mr. Haskins, okay? I want you to assume -- first of all, I think I misspoke. I want you to assume that the work he was doing was on bonnet gaskets, not that the type of material would matter to a doctor, but assume that the gaskets were adhered to the bonnets on Buffalo pumps. Can you make that assumption?

A. Okay.

Q. In a person -- in Mr. Haskins case, where he was exposed to asbestos from the Buffalo pumps bonnet gaskets, as well as other sources of asbestos, do you conclude what caused his mesothelioma?

A. Can you say that again?

Q. Yeah. Mr. -- going back to Mr. Haskins.

A. Okay.

Q. You've got the gaskets exposure from working with the Buffalo pumps plus other asbestos such as insulation on the ship. In that -- based on that scenario, which is what the facts we believe we will prove at trial, what caused Mr. Haskins' mesothelioma?

A. I'm assuming that you are including all of Mr. Haskins'

exposure.

Q. Correct. Correct. It's everything.

A. Right. Each of the exposures were a contributor to the sum total that makes up the cumulative dose.

Q. Was the gasket exposure medically significant to you?

A. Yes.

Q. And did you believe it was a contributing cause of his mesothelioma in a medically significant way?

A. Yes, it was.

Q. Is "substantial contributing factor" a medical term, or is that a term that you were asked about in courtrooms like this one in front of juries?

A. It's more of a legal term.

Q. Okay. If I were to tell you or ask you to say -- what I mean by "substantial" is it makes a real and important contribution to the medical situation that developed in Mr. Haskins. It was not trivial and not hypothetical, but it was significant and important. Was the exposure to asbestos from working to remove gaskets from Buffalo pumps equipment that were on the bonnets of Buffalo pumps equipment for three months a substantial contributing cause of Mr. Haskins' mesothelioma?

A. Yes. Or medically was a significant contributing factor.

Q. Okay. Have the opinions -- to the extent you've offered opinions here today, have your opinions been based to a

reasonable degree of medical certainty?

A. Yes, they were.

Q. And have you discussed the bases and the methodology you followed in reaching your causation conclusions in the Haskins' case?

A. Yes, I have discussed here, and I have discussed them in my report.

MR. FINCH: Okay. And, Your Honor, at this point, I would like to turn the questioning over to Mr. George.

THE COURT: Sure.

MR. FINCH: I don't believe he's going to have extensive questioning, but you never know.

MR. GEORGE: Very short.

THE COURT: No problem.

DIRECT EXAMINATION

BY MR. GEORGE:

Q. Dr. Bedrossian, I want to turn your attention to the Cheshire case, and that's a case that you did a report in February 12th of 2016. Do you recall that?

A. Yes, I do.

Q. And at the time you prepared your report, you had looked at the medical records, you had looked at the pathology, and you had some understanding of his occupational history, correct?

A. Correct.

Q. Before you submitted that report to the defense, did you do anything else to determine what Mr. Cheshire's occupational history consisted of?

A. Yes. I reviewed additional materials that I received.

Q. And was one of the additional materials his sworn testimony?

A. Correct, his deposition.

Q. And what did you find out in reviewing that testimony about the nature of Mr. Cheshire's exposures to asbestos?

A. That he did have the exposures, that they were frequent, they were close, he was in the immediate area, and he did that repeatedly.

Q. Let me offer you a hypothetical. I want you to assume that Mr. Cheshire was in the United States Navy from 1965 to 1989. I want you to further assume that from March of 1966 to March of 1967, he served aboard the USS *Cadmus*, C-a-d-m-u-s, a repair ship, assigned exclusively to the valve repair shop where he routinely and regularly removed and replaced asbestos-containing gaskets and packing from various valves, including Crane valves. I want you to further assume that in performing that work, he used a scraper and wire brush to remove remnants of the used asbestos gaskets from the bonnet surface of the valve, including Crane valves. And that was a process that created visible dust, dust that Mr. Cheshire breathed.

Further assume that when he was stationed aboard the next ship, the USS *Henderson*, the ship was in dry dock for three to four months at Mare Island for an overhaul, and during that overhaul period, he was again assigned to the valve repair shop where he did the same type of activities he did when he was on the *Cadmus*. Further assume for the remainder of his Naval career, he worked with and around individuals that used asbestos-containing products, but his hands-on work was more limited at the tail end of his career than it was in the beginning.

Based on that totality of assumed facts, do you have an opinion as to the cause of Mr. Cheshire's mesothelioma?

A. Yes.

Q. And what is that opinion?

A. The opinion is that the totality of the exposures that he had, whether they came from the gaskets or they came from the ship that has insulation, all of them were parts of the sum total of his cumulative exposure.

Q. And what does that -- what type of methodology did you use to arrive at that conclusion regarding causation?

A. Um, I think I outlined them already. But essentially I did a medical review which is based on the differential diagnosis concept, taking into account the general causation that has been established in the case plus the individual factors that entered my reasoning.

Q. When you perform that type of analysis, do you take into consideration the different fiber types that are in the different types of products?

A. Yes, I do.

Q. And do you take into consideration the extent, the duration, and the proximity of Mr. Cheshire's exposure to those products that are generating airborne dust?

A. Yes, I do.

Q. I want to adopt a similar type of hypothetical that Mr. Finch gave. I want you to assume patient A, patient A has been properly diagnosed with a malignant mesothelioma; and when his exposure history was investigated, it turns out that he spent about a year's time aboard one ship in a valve repair shop where he routinely and regularly removed asbestos-containing gaskets from the bonnet of the valve, some of which were crane, some of which were other materials, and that he did it again on another ship for a three- or four-month period.

If you were given those exposure facts for a given patient that had mesothelioma, what could you conclude about the causation of that disease?

A. That his cumulative dose to the exposure to the two circumstances that you described added up to a cumulative dose ultimately responsible for his mesothelioma.

Q. And so was the exposure contained in that hypothetical

sufficient in and of itself to cause disease in certain individuals?

A. Correct. You know, with the caveat said that there were intense, frequent, and repeated.

Q. Given the fact that Mr. Cheshire's exposure began in the Navy in 1965 and he was diagnosed in 2015, was there sufficient latency period to count those exposures as causative?

A. Yes, there was.

MR. GEORGE: That's all I have, Your Honor. Thank you very much.

THE COURT: Yes, sir, Mr. Finch?

MR. FINCH: Your Honor, we would pass the witness for cross-examination, given the -- we reserve the right to redirect. I'm not sure one or both of us. Pass for cross.

MR. MERIWETHER: Your Honor, I should rise when I speak.

THE COURT: Say that again.

MR. MERIWETHER: I should rise when I speak.

Your Honor, Robert Meriwether on behalf of Air & Liquid Systems. We want to accommodate the Court's schedule. We want -- I notice -- I know we are hitting close to 12:30.

THE COURT: Does that mean you want to start at 1:30?

MR. MERIWETHER: That is perfect. I mean --

THE COURT: Perfect. That's all we ask for here.

MR. MERIWETHER: We can go -- perfection is all we have as to offer.

THE COURT: That is all we can give you. All right? We'll start again at 1:30.

(Thereupon, there was a lunch recess.)

MR. MERIWETHER: Might I, Your Honor?

THE COURT: Yes, sir. Robert Meriwether on behalf of -- I think I have drawn the short or long straw. I get to go next. So with your permission, I will come up to the spot that was previously occupied by opposing counsel.

CROSS-EXAMINATION

BY MR. MERIWETHER:

Q. Good afternoon, Dr. Bedrossian.

A. Good afternoon.

Q. It's nice to see you again. I want to touch on a number of things that you talked about when you were talking with your -- with my opposite member who represent the plaintiffs in these cases and ask you about some of your statements to this Court. Let's just start with the simple fact that you have stated in the past that exposures -- let's get the precise amount. You have stated in the past, haven't you, Dr. Bedrossian, that exposures of three weeks' duration to airborne asbestos from Thermal Systems insulation were not enough to cause mesothelioma. Is that not correct?

A. Can you repeat that?

Q. Sure. You were questioned about an exposure to an individual who for three weeks was breathing asbestos insulation dust from pipe insulation and you said that you had not seen mesotheliomas due to just a three-week exposure. Isn't that correct?

A. I may have said that, but that doesn't mean that I believe that three weeks is not sufficient. What I said is I haven't seen it.

Q. I see. All right.

Let's talk for a moment about one of the citations that you have in your report. Well, strike that. I'll try to do a preliminary first, so it's a little bit more sequential, a little bit more logical. In this case, in Mr. Haskins' case, which is the one that I'm discussing today, although it may apply to the Cheshire case as well, you haven't performed any calculations, formed any opinions about the cumulative dose of asbestos that Mr. Haskins might have received from working with any specific defendant's product, correct?

A. Correct. And the reason I didn't is because it's not necessary.

Q. All right. It's not necessary for you to know that dose, I understand. You've not attempted to quantify the release of dust from any defendant's product. You have no

information on the release of dust from any product that Mr. Haskins worked on, correct?

A. Well, I have information from the literature, from Dr. Millette and other studies, that gaskets do release fibers to a considerable concentration.

Q. All right. We'll come back to that. So you have from Dr. Millette an estimate of what you believe gaskets can release, correct?

A. Correct.

Q. We'll come back to that. You are not able, as you sit here today, to quantify the exposure that Mr. Haskins may have received from any Buffalo pump, correct?

A. No, I don't. Primarily because I don't need that information.

Q. You don't need it. I understand.

When you were talking with plaintiffs' attorney, there was a good deal of discussion about what amount of asbestos exposure is necessary in order to cause mesothelioma, and there were a number of arguments that were advanced by plaintiffs' counsel, and to which you agreed, that tended to bear on the issue of how much particular asbestos fibers were required to cause mesothelioma.

Now, among those were discussions of fiber type, that is, does it take more of one fiber type than another to cause it and the concentration of fibers that get to the

lung. So let's take these things one by one.

One of the claims that you made on the stand this morning was that chrysotile breaks when it hits a split in the airway and that the resulting piece can be straight. Where is the scientific study or studies that indicate that?

A. There are several studies where the fiber utilized was chrysotile. And I know by experience that when you examine the lung of a patient, the chrysotile doesn't stay intact with all its curves and bends and circulations. They appear as short fibers, around five micro in diameter, I mean in length.

Q. Let me get this straight. You believe because you have seen short chrysotile fibers in the lungs of a deceased individual that you can then assume that those fibers were long when they were breathed in and that they broke up when they hit places where the airway splits. This is your assumption?

A. No, it's not my assumption, it's my belief because there are other sorts of studies that do the same. You can do an analysis of the fibers looking at them by electromicroscopy and you can do a spectography of the fiber and it has the same composition whether it's short or long.

Q. All right, sir. Can you point me to a single study, published or unpublished, which would tell me the degree of force that is necessary to break a chrysotile fiber, not

longitudinally, but along its width access?

A. I don't think there is a study of that.

Q. I didn't think so. Can you give me a single study, published or otherwise, which indicates the force that is exerted on a fiber when it impacts a split in the airways as it is breathed into the human body?

A. I'm not familiar with any studies measuring the force. I don't even know what unit of force you would be referring to. But I don't need to know the mechanism to know when I find something breaking in two, like I can conclude that some force was applied.

Q. So because you can conclude that some force was applied at some point in that fiber's life, it is your presumption that that force was the impact of the inhaled fiber on the tissues of the body as the airway splits?

A. If I don't see imprints on it, I can assume that it was broken and not bitten.

Q. Okay. One of the interesting things that you mentioned at the beginning of your discussion with plaintiffs' counsel was that fibers translocate via the lymphatic system to all areas of the body, including the eyeball. And that they were a systemic carcinogen. Can you tell me whether asbestos is associated with eyeball cancers?

A. I didn't say that every area that it got to, you got the cancer. But individual cases, you have kidney cancer,

thyroid cancers, GI cancers, esophogeal cancers.

Q. Is it your belief that kidney and thyroid cancers are associated with asbestos exposure?

A. Epidemiologically kidney for sure. When I say that is a systemic carcinogen, I'm saying it is known as a carcinogen. Since it is distributed systematically, therefore, is a systemic carcinogen.

Q. I see. It's because it reaches that area of the body that it must be a carcinogen there, not because of there is any epidemiological evidence to support that?

A. There is a lot of epidemiological evidence of nonpulmonary mesotheliomas.

Q. Listen carefully to the question, Doctor. I understand that there are distinctions in mesotheliomas in the mesothelial areas in the body that aren't pulmonary. My question had nothing to do with that. I am asking you whether or not your description of this fiber, this asbestos fiber as a systemic carcinogen, which you said it was, and you said it was translocated to all areas of the body, including the eyeball, and you just told me that you believed that thyroid cancers were associated with asbestos. So let me ask you one more time. Do you believe that thyroid cancers or ocular cancers are related to asbestos exposure?

A. No, because I never said that.

Q. Okay.

A. What I told you, I defined for you the basis for my statement. Asbestos is a carcinogen. It is recognized as such by many agencies and by the scientific community. Asbestos fibers are also distributed systematically, and I said that explains the number of case reports and case series of cancers in those organs. I cannot say in every organ or in every instance.

Q. All right. I believe you also said this morning that if you have exposure to asbestos and you get malignant mesothelioma, then the mesothelioma was caused by an asbestos until proven otherwise?

A. Right.

Q. Do you recall saying that?

A. Yes.

Q. All right. So if you've got malignant mesothelioma in a patient and there was some exposure to asbestos, it is not necessary to look and see if there is any thorotrast, therapeutic radiation, aeronautic exposure in Turkey, long history of empyema, of the other causes, you don't need to look for those?

A. You may have misunderstand when I answered. I said very clearly that once you make that assumption before you accept it as scientifically correct, you do a differential environmental consideration of alternatives; and among them, I mentioned radiation, thorotrast and empyema. Obviously you

have to rule them out, which I did in both cases.

Q. All right, Doctor. Let's drop back for a minute and leave aside some of the statements that you made in your testimony this morning and talk a little bit more generally about the subject which is mesothelioma and causation.

To begin with, I think it's no surprise to this Court that mesothelioma is a very rare disease. There are approximately 2,000 to 3,000 cases of mesothelioma in the U.S. every year, correct?

A. Yes. It varies a little bit, 3,000, depending on the person who wrote it.

Q. And as you described, not every mesothelioma is caused by asbestos, correct?

A. Right. With the caveat that the vast majority, the overwhelming majority is.

Q. Do you have an estimate as to the number of, or the percentage of mesotheliomas that are caused by asbestos exposure in America versus the percentage that are not? You may break it down by gender, if you wish.

A. Um, it depends on the tool that you use to determine that fact. If you base it on death certificates, there will be minimum. If you base it on medical -- known pulmonary specialists taking a history would be also low. But when you take it, properly obtain history with someone trained in taking occupational and environmental histories, is upwards

of 90 percent.

Q. All right.

A. And the British study was as high as 96 percent.

Q. You would agree that according to some studies, some 10 to 20 percent of mesotheliomas in men are not related to asbestos?

A. No.

Q. You wouldn't agree that that is true in some studies?

A. No. What they say is the cause of the asbestos exposure is not determined, is not known. The fact that it's not known, it doesn't mean that it did not exist. There are several studies where, for example, in Australia, they determined that 90 percent of cases were not, but then when they look at the cases that there was no evidence of that, and they retook the exposure history and examined the tissues for asbestos, they found that those individuals, although they thought they didn't have exposure, that they did have exposure.

Q. So when we cut to the chase, do you have a number of asbestos-caused mesotheliomas versus nonasbestos caused mesotheliomas in men? Do you have that number?

A. Well, it's the vast majority in some.

Q. I understand that, but I was looking for a number.

A. Some articles may say 80, and another may say 95, but I explained to you that that number is not to be taken

literally. There is evidence that that number is erroneous because the patient may forget that he was exposed, the history may not be taken correctly, the data may derive from death certificates or the data was not accompanied by measuring the asbestos in the tissue.

Q. All right. Though it's not at issue in this case, let me ask you: Do you have a number for the percentage of mesotheliomas in women that are not caused by asbestos?

A. Um, the same answer applies, with the caveat that in women, because it was believed that occupational exposure are primarily in males, they overlooked the fact that that person may have had exposure through a different mechanism. They may have lived near a factory that used asbestos. They may have lived in a highly contaminated area, like under a freeway in a major city, or some of their relatives, either brother or the father, brought home asbestos in their clothing.

Q. All right, sir. Let's skip to something else that you mentioned in your testimony. You talked about mesothelioma being a dose-response disease, correct?

A. Right.

Q. The higher the dose, the greater the risk, correct?

A. Right.

Q. This is the same concept that people refer to as the dose makes the poison, right?

A. I'm sorry, what?
Q. The dose makes the poison. It's a toxicologist statement.
A. To extrapolate from that is full of, you know, possibilities of not connecting the two.
Q. I'm not asking you to extrapolate. I'm just saying this is the concept, that the greater the dose, the greater the risk, correct?
A. If you are saying the greater the dose, the greater the cumulative dose of the carcinogenic component of the poison, yes.
Q. Now, you do agree, and you've agreed in the past, that the background level of asbestos, the ambient asbestos level in the air, which varies considerably obviously across the United States, but that ambient level does not cause disease, correct?
A. Correct. You know, with the exception we already discussed. In regional areas, it would be higher than, for example, in the field, in the site.
Q. Of course what that ambient air is in the countryside would be affected by whether or not there are natural outcroppings of asbestos-formed minerals in that area, right?
A. Right. That is what I just said.
Q. So you agree that the exposures that we all have to ambient air would involve millions of asbestos fibers over a

lifetime, correct?

A. Um, I don't know if that would be true in every case, every person in every location.

Q. But averaged across the population, it would be true?

A. I think early on I indicated that an occupational exposure -- exposed patient accumulates billions of fibers in the same period of time that a person exposed to air with the normal concentration, which is less than .0005 accumulate fibers. So the difference between occupational exposure and ambient exposure.

Q. That is a lovely answer to a question I did not ask.

Let's return to what I asked, which is that you would agree that over a lifetime, the exposure to established levels of ambient asbestos, background levels of asbestos, would involve exposure to millions of asbestos fibers, correct?

A. Right.

Q. Do you have a median level that you used to calculate that background level of asbestos? Is there something that you use?

A. No, I do not use it.

Q. All right. So when you say that, and you agreed with me a moment ago, that background exposure does not cause disease, what are you talking about if you don't have a level?

A. Because it's not necessary to quantitate the exposure. It's not that the ambient level does not cause mesothelioma, because if it did, mesothelioma would not be a very rare disease, it would have occurred at least in some of the people who are sitting in this room.

Q. Right. Because if ambient levels caused it, there would be millions of cases?

A. No. No. In the ambient level, there is not going to be millions of fibers, and therefore millions of cases.

Q. Okay. You've lost me now. I thought you just told me that you don't need to know the level of ambient exposure in the air because you know that there are not an excess of mesotheliomas in the population that is just exposed to ambient air, correct?

A. Correct.

Q. And if you extrapolate from the number of people in this room to the population of the United States, and you say that there would be people in this room with mesothelioma if ambient exposures caused it --

A. I said if ambient exposure was in the billions.

Q. Was what?

A. In the millions or billions, for a total life span.

Q. I see. So you simply disagree with the calculations of how many fibers a lifetime of exposure to ambient asbestos levels. You disagree with the calculations there, that is

fine. Then we'll move right on.

You do not have a background level that you cite. Do you have a calculation that you would use to estimate the exposure to work on a gasket, such as Mr. Haskins allegedly did on gaskets associated with Buffalo pumps? Do you have a level for that?

A. For ambient air I used the figure that Dr. Wagner uses. For asbestos exposure in a gasket person, I'm not an industrial hygienist or a material scientist or an occupational physician, so I don't need to measure that.

Q. Okay.

A. Because I know by experience that other individuals doing similar work in work studies and other type of studies have shown that they do get exposure to asbestos that contributes to the total cumulative dose.

Q. All right. So -- and I think you just told me that you used Dr. Roggli's numbers for ambient background levels of exposure to asbestos. Is that what you just told me?

A. What was the first part of the question?

Q. No. I'm asking you what you said, not what I said.

A. Yeah.

Q. Didn't you just tell me that you --

A. That I said --

Q. I think you just told me that you used Dr. Roggli's estimates for background or ambient asbestos exposure. Is

that correct?

A. Correct.

Q. Okay.

A. But --

Q. What is that number that Dr. Roggli uses?

A. The number is unimportant.

Q. No, I'm asking you what it is. You said you use it.

A. Well, I already answered that. So go back and look it up.

Q. Okay. What is the number that Dr. Roggli uses that you say you use? Do you know it?

A. 0.000005.

Q. 0.000 --

A. What's important is the power of the difference, in other words, is thousands of times greater.

Q. Okay. Well, finally, given that you use 0, and now you have one, two, three, four zeros and a five, if I am correct, what you are now telling me is that 0.0005 fibers per cc over a lifetime does not cause disease. Is that correct?

A. Yes, but --

Q. Okay. Fine.

A. You are implying that I used that figure and I'm telling you that I don't deal in numbers. It suffices for me to know that four to the fifth power of the difference.

Q. Okay.

A. Measuring the exact amount either in the ambient air or in the patient working with gasket is not important. I'm not an industrial hygienist who make calculations and use that for my conclusions.

Q. In addition to the 0.00005 estimate for background, you know that there are other estimates that range from 0001 and down, correct?

A. I know that there are other numbers. The one that sticks in my mind, in Europe they use 0.0003. And I'm sure different studies would have numbers all over the place because they are rather inaccurate measurements.

Q. So the European number, do you believe that 0.0003, do you believe that that number causes disease?

A. No.

Q. Fine.

A. That just a number of ambient air.

Q. Exactly. So now we have two numbers that you believe do not cause disease. Do you have any number that you would attribute to Mr. Haskins' exposure to asbestos in connection with working on Buffalo pumps?

A. I don't have any number. I don't deal in quantitation.

Q. Don't need that. Okay.

Let's talk a little bit about what happens when you breathe in an asbestos fiber from any source, because the body doesn't know the difference between a fiber that it got

in the ambient air and a fiber that it got in the workplace, right?

A. Well, except that when he gets truckloads of it relentlessly, overwhelmingly coming down the lung, one sure recognizes an overwhelming exposure, as opposed to a minuscule concentration in the ambient air.

Q. Let's talk about those defense mechanisms. You mentioned some of those this morning. You've got the nose hair, which filter out fibers if you are breathing through your nose. You've got those bins in the airway, curves where fibers tend to stick to the saliva or mucus covered walls of the airway passages all the way down to the lungs?

A. You have the mucociliary. They don't necessarily need to be curved. They are more like straight but bifurcated.

Q. Right. You've got the mucociliary escalator, which has all the little cilia, the little hair-like, finger-like appendages on the cells that beat upwards, moving a curtain or a sheath or a covering of mucus and all of the crud that sticks to it, it comes up and you either swallow it or spit it out, right?

A. Yeah. But that capacity is not unlimited. That is same for to say since a team has a goalie, there would be no goals to score on it. Because the defense is there, it doesn't mean that it's not overwhelmed and overtaken.

Q. And the lymphatic system that you described earlier as

being part of the translocation of fibers, that lymphatic system can also carry fibers out of the body entirely to be excreted, right?

A. Depends on how far you want to go. Eventually the fibers that did not get digested by the macrophages, the ones that did not break into smaller and smaller pieces, the residue at the end will gain access to the circulation and filter in the kidney and be expelled as urine or present in urine.

Q. Understood. I believe that you have confirmed for me in the past that the defense mechanisms of the body are sufficiently efficient that 90 to 95 percent of most of the dust we encounter is eliminated or removed from the body, right?

A. I don't believe I said that.

Q. All right. That's fine. Do you agree with it?

A. No.

Q. Fine. How much of the dust that we ordinarily encounter is removed from the body by the defense mechanisms?

A. I don't believe there have been a study with quantitation of that level. Primarily because there is no apparatus that you can put near the mouth or the nose of the person that reliably measure that amount. That amount is estimated by air sample devices that measure the number of bits of the particular, but there is no accurate and infallible instrumentation to estimate the real amount of asbestos that

the person inhales.

Q. So in this case you need some accurate and infallible instrumentation to give precise numbers. So that is a place where you do need that kind of precision?

A. No, I didn't say that. I said if I were to do a measurement, that is what the conditions apply to that exercise. I said very clearly that I don't need to measure the concentration at the point of entry or during inhalation, neither I need to measure concentrations of what remains in the lung or the pleura.

Q. Let's talk about another way that exposure can work. Before you even get to these defense mechanisms that we have been talking about, you talked about the concept, or at least you alluded to it, I don't know if you used the term, laminar air flow, where the fiber lines up with the airwaves and lines up with the air flow, and there are certain fibers that you will breathe in and you will breathe right back out because of --

A. There is no such thing.

Q. So for you every single fiber, if you say there is no such thing, that is fine, Doctor.

A. Yeah, but I need to explain it because you interrupted me.

Q. Please do. And I beg your pardon.

A. A fiber has mass, therefore, is subject to gravitational

forces. There is no way that a fiber that is not alive doesn't have movement of its own. It comes down the lung this way and passes through all the bifurcations and has all the impact and then stays there, impacts and somehow the pleura pushes it exactly the same way that it came in. It's full of that dust, and as it will break and get to the alveoli, it becomes trapped there because it's not going to find the opening to the bronchi and line it up perfectly. That would have been a live pleura with a central nervous system to calculate --

Q. All right.

A. -- what it needs to do.

Q. I think I understand the gist of your point, which is I believe you are telling me that no single fiber that anyone inhales comes back out again. It is always going to impact something and stick or be processed. Is that what you are saying?

A. Qualitative words in there, "always," and what I said is this is what scientifically happens.

Q. All right. Well, let me ask the question again, because I thought that is what I was getting at. Doctor, some of the fibers that you breathe in travel in and travel back out on air flow without impacting the body, isn't that true?

A. No, it's not.

Q. Okay. Then the reverse of that must be true, every fiber

that you breathe in must impact the body in some way, none of them are breathed in and breathed back out?

A. Correct.

Q. Okay. Fine. So every fiber you breathe in impacts the body in some way. That includes background fibers, that includes single fibers from very minimal exposures that literally only generate a single fiber, but if you breathe it in, it impacts the body, correct?

A. The defense of the lung is the same regardless of the I.D. tab or the identification tag that a fiber might have. Obviously, don't have it, and therefore the lung treats them equally. An equal opportunity defender.

Q. All right. Let's talk about some of what you said about cancer causation, mesothelioma causation this morning. One of the citations that you made in your report and that you discussed in your testimony this morning was, the title is sort of a mouthful, people were calling Iwatsubo, but, "Pleural mesotheliomas dose response relation at low levels of asbestos exposure"?

A. What was the first author?

Q. Iwatsubo?

A. How do you spell that?

Q. I W A T S U B O?

A. Iwatsubo. Okay.

Q. You cited to Iwatsubo on page 5 of your report for the

proposition that, "The carcinogenic effect of asbestos is cumulative regardless of the source of exposure and the total dose has been consistently found to be the best indicator of risk and dose responses best indicated by cumulative exposure index." I want to ask you, do you have a copy of Iwatsubo there or are you familiar enough with the article that you cited? Do you need me to get you a copy?

A. You can get me a copy. It would be easier.

MR. MERIWETHER: May I approach?

THE COURT: Sure.

MR. MERIWETHER: Here you go, Doctor. I did get an extra copy in case you wanted the paper paper.

Q. On the next to the last page of that article, Doctor, which is page 141, on the left-hand column and the first full paragraph, it begins "some indication"?

A. You are talking about page 140?

Q. No, I am talking about the next to the last page of the article, page 141, left-hand column, the first full paragraph beginning with the words "some indication".

A. Right. That is the end page of the article.

Q. Okay. So are you with me?

A. Yeah. Yes.

Q. Okay. In that paragraph, despite your citation to it, what it actually says is, "There were no cases of mesothelioma among members of the cohort of Australian blue

asbestos workers who were exposed for less than three months. None among the North American insulators whose exposure lasted less than 15 months. And only one, rather than the 25 expected, among Rochdale textile workers exposed for less than ten years. These cohorts do not, however, provide data that allow us to examine the effect of low intensity exposure." That is what it says?

A. Yes. That's why I told you that isolated studies may deviate from the norm, but the consensus is that cumulative exposure is the cause of mesothelioma.

Q. Let's talk about some of the isolated studies. You just told me that isolated studies without larger consensus can be used to form your opinion, right?

A. Right, but --

Q. Let's talk about some of the isolated studies that you cited this morning. A 1967 case report where an individual mixed asbestos cement for use in his home. Do you remember that?

A. Yes.

Q. All right, sir. And I believe that you said that he was mixing raw asbestos into cement to make his own asbestos cement, correct?

A. Right.

Q. So that means he had bags, or bag, or a bag of raw asbestos fiber in his basement, right?

A. Yes.

Q. And based on that, you believe that that was sufficient to get him a dose of asbestos that would -- that caused his malignant mesothelioma, right?

A. That was the conclusion of the author of that paper. But let me go back to Iwatsubo. You did not emphasize the last sentence of that paragraph. He said, to describe what you just told us, he said clearly, "These cohorts do not, however, provide data that allow us to examine the effect of low intensity exposure." The fact that you observe that did not enter his determination whether or not low exposure causes mesothelioma. These are just observations that he made.

Q. I think I did read that. I meant to.

A. But I don't think you --

Q. My point is that those are not enough to determine that low level exposures cause --

A. No. It says that are not data that allow to examine that issue. So it's observation, but it's not the type of observation that allows him to have the deductive scientific approach to answer that question.

Q. And believe me, Doctor, what I am emphasizing today is just that deductive scientific approach. So I appreciate your pointing that out.

Let me ask you to move on, then, if you would. Is

there anything in the 1967 case report about asbestos cement use, Doctor? Doctor? Is there anything in the 1967 case report about the man who mixed asbestos cement in his basement to indicate what fiber type is in the cement?

A. Well, what is the name of the -- did you say an author?

Q. No. All right. Would it help if I retrieved the Iwatsubo article? It seems to be distracting you.

A. No, I need it.

Q. Doctor, in the 1967 case report where an individual mixed asbestos cement in his basement, is there anything in there to indicate what fiber type of asbestos was used in that cement?

A. That is where I said I need to look at the specific article.

Q. If you know, fine; if you don't, that's also fine.

A. No, I don't at this moment. If I look at the article, I'll be able to answer.

Q. That's fine. On direct examination this morning you said that the asbestos was chrysotile. My answer -- my question for you is how do you know that? You think it's in the article?

A. Well, as I said, it is my assumption, but in order to verify it or not, I need to look at the article.

Q. And if your assumption is that it was chrysotile, and your assumption is that he had raw fiber in his home to mix

into the cement, is it also your assumption that he was exposed to that same raw fiber every day thereafter?

A. I can't address that. You are giving me a hypothetical.

Q. It's not enough. I understand.

A. And I'm not a scientist that deals in suppositions.

Q. Another case report which you mentioned this morning was a case study where a man sawed asbestos boards for use in building a shed. May I ask you the same question? Do you know what fiber was in that board?

A. No. It would help if you give the article so I can look at it.

Q. Doctor, I don't have the article up here, you are the one who cited it. I'm just asking you a question. Do you know what fiber was in the board?

A. I need to look at the article.

Q. If you don't know, that's fine. Do you know whether he continued to use and be exposed to the asbestos board shed thereafter?

A. You are asking me for details exactly on the route that the author took to get to an end point. What is important is the end point. The exact route, which measurement he did, which fact he considered, are in the article and speak for themselves.

Q. I understand. So Doctor? Doctor?

A. Yeah.

Q. If you are citing articles that are case reports for the proposition that brief exposure can cause mesothelioma, don't you need to know the details of that exposure?

A. I do, and they are contained here, but you are not giving me a specific article to answer a specific question. The importance of the articles is that they established the precedent that such exposure occurs. The evidence for such assumption or such conclusion comes not only from the case reports that I might have cited here, or the case series, they also come from epidemiological studies, including all the other factors that I told you in the holistic approach to determine causation. You are trying to specify to a very specific portion of this article as if that is the one that makes or breaks the conclusion.

Q. All right, sir. Let's drop back for a moment and talk about the ways that these small doses of asbestos, these limited exposures cause the disease really at a molecular level. And you touched on this briefly this morning. I want to explore it a little bit more. Most mesotheliomas are monoclonal, in the sense that they start from a single cell and there are repetitive injuries causing genetic errors in that cell or its progeny, it's daughter cells, correct?

A. There is evidence for that. But more recently, there is evidence that you have multiple clones, whereby a genetic alteration caused by the first impact creates a gene which

itself may not transform that cell but may affect a separate cell and create a second clone.

Q. So if you are looking for the way that asbestos can cause these changes, the most likely event -- and I understand that we are in the realm of theory here because nobody has tested this -- but the most likely event is the asbestos fiber either contacts that cell which eventually turns into a mesothelioma or contacts cells in the immediate area which then, through signaling, cause changes in that cell, correct?

A. I cannot pinpoint which cell. That is --

Q. I understand.

A. -- the reason why we use the term cumulative. It may hit that cell and it may create gene, but down the line, the defense mechanism may splice or cut off the sequence that is carcinogenic; and therefore, that particular cell did not participate in the process. But over many years, with 16 breaths per minute with high amounts of asbestos, eventually the correct sequence of events that end up being cancer occur as a result of the cumulative effect.

Q. Understood. For me, one of the most helpful ways of looking at that complex process is in that Dail and Hammar book that you have cited to several times. This is at page 598 of that book.

A. Um-hum.

Q. It indicates at the very top, the beginning, you have the

mesothelial cells, you've got the impact of asbestos fibers, you've got the potential creation of reactive oxygen species, reactive nitrogen species, genetic mutations. Of course as you go through this branching tree to eventually get to a fully malignant mesothelioma, there are a number of situations in which that cell is killed off either by the body's natural defenses or some other mechanism. And so there are a number of things that happen over the course of many years to move from this point at the top where you've got an original mesothelial cell to this point at the bottom where you've got a mesothelioma, correct?

A. Correct. But the intermediate portion of your explanation you said that the mesothelioma cell may be killed off. Well, that would be one of the events that happened to protect the patient. But it's known that asbestos causes a cell death invitro, but in human beings as a result of this genetic transformation, the cell is immortalized, it continues multiplying, and therefore participates in the development of the cancer.

Q. I think we are saying the same thing, which is that in order to become a fully malignant mesothelioma, this cell line has to escape such things as a apoptosis cell death, programmed, planned cell death. It has to figure out a way around it. And like all cancers, it needs to make sure that the body doesn't kill it off in order to survive, and that is

what mesothelioma does, correct?

A. Yes. But my answer was addressed to the fact that asbestos causes cell death. And I wanted to make sure that you understood that death phenomenon is very, very improbable or doesn't occur that often. Most likely event from that is that the genetic changes amount to immortalization of the cell.

Q. When you talk about this kind of impact on a single cell or its progeny, its offspring, its daughter cells, and you can see on this chart the points at which asbestos may be contributing to that process. On the chart, the asbestos fibers are blue, perhaps the reference to crocidolite, which is the blue African asbestos, perhaps that is the color the printer had, but when you look at those points, it is a complex process. And you would agree with me that we cannot figure out which exposure over time actually made those changes to that cell. There is no way to go back and figure that out?

A. Exactly. You are saying what I would have liked to have said.

Q. And so once we are in that position, what we are trying to do in order to determine cause is to figure out whether or not there is scientific evidence that an amount of asbestos could cause these changes; not whether it did?

A. No. The methodology I already explained is a holistic

approach. Now we are talking about one aspect, this addresses the plausibility that asbestos can be a cause of mesothelioma. We are talking about genetic events, but this entire constellation, all the way from the exposure, all the way from effects of asbestos in the body, like pleural plaques and asbestosis, all the way to the pathogenesis, which is different than carcinogenesis, and the fact is that they repeatedly put the asbestos in this picture just to remind you that it affects not only the beginning of the process, but steps down the line until the final transformation of the cancer cell.

Q. I am not sure that you understood my question. Let me try again.

Since you've agreed with me that we cannot retroactively figure out which exposures to asbestos, which fibers caused the changes, some of which are shown on this chart, that led to an individual's mesothelioma, since we cannot retroactively figure that out, in order to determine causation, what scientists have to do is to figure out whether someone was exposed to enough asbestos that it could cause the mesothelioma, not because it's impossible, figure out what exposures he had that did cause. Do you understand my point?

A. Well, taking into account that what you are saying is very confusing, the fact is you cannot pinpoint a fiber. You

cannot pinpoint the exposure. Neither can you exonerate them. And that is the reason why the scientific evidence speaks of cumulative dose. We can say that the different exposures contribute to the total and we can say that the total is responsible for the complex sequence of events that end up in cancer, but we cannot blame one fiber. We cannot blame one event. We can never find which cell was the initiator because it takes multiple changes that are in a sequence and they have to be all lined up for the person to have the cancer. That is the reason why when you have exposed populations, only ten percent of them will obviously be exposed to asbestos, and the other ones you need to look for other explanations.

On top of that, you have to place the individual susceptibility. What does that mean? It means that the person, each person has a different system, like one person may be incapable of stopping apoptosis, so therefore, that person is immune to the cancer, because when you knock out apoptosis, it comes back and kills the cell and avoids the immortality contribution.

Q. All right, sir. Let me circle back to one thing you said in that fairly lengthy answer, where you told me that it's because you cannot exonerate any exposure that you must consider them all cumulatively, correct?

A. I didn't say it was because of that. When I said that is

because of the entire body of evidence, all the way from clinical to molecular.

Q. We'll dig that out for you, Doctor.

So it's your position currently, from what you've told me so far, that every asbestos fiber that you breathe in impacts the body in some way because none of them are breathed right back out, that you cannot figure out which of those fibers caused the mesothelioma; that is, you can't exonerate any of them, so you consider them all cumulatively. And what I want to ask you about now is some of the differences -- well, here. Let me hand you a copy of the deposition that you gave in this case. It's got all your exhibits there. If you turn to page 31. When you are talking about cumulative dose --

A. Okay.

Q. -- you are actually being cross-examined in this case at this deposition out of yet another deposition which you gave but fortuitously, I believe it is in the companion case that you are here for today, that is Cheshire. So you are being asked in the Haskins deposition about the following, you say, "I cannot pinpoint one exposure or exonerate another. So that is why our conclusion is the total dose that the patient underwent from each product that contained asbestos that were in his surroundings where he worked for a long period of time, repeatedly in close proximity to it, those exposures to

that product contributed to the total cumulative dose and it is the total cumulative dose that is the cause of the mesothelioma." Correct?

A. Correct. But you are not understanding what the meaning of it is. It is not cause because of a negative finding that I'm unable to pinpoint or exonerate, it is the fact that what leads to that conclusion are the investigations that I did all the way from history, all the way to genetic materials. So maybe was an important use of the word why, but you were trying to imply that this here is the entire basis for my conclusion, and that is incorrect. And for that reason today I laid out the entire sequence of reasonings that are important to determine the contribution of the asbestos exposure.

Q. Let's talk for a moment -- those are the only questions I have out of that deposition for right now.

Let's talk for a moment about the different kinds of asbestos. I believe that you confirmed for plaintiffs' counsel this morning that you believe that the gaskets at issue in this case contained chrysotile, correct?

A. Correct.

Q. Now, obviously you went through and distinguished between chrysotile and the amphiboles, correct?

A. Correct.

Q. And chrysotiles are serpentine, amphiboles, which include

the commercial others of crocidolite and amosite, they are stronger, straighter, pointier, so to speak?

A. Right.

Q. They -- these different fiber types have different chemical makeups. They have different morphology, different shape. They have different electrical properties. They are just very different things, correct?

A. Correct.

Q. And you have confirmed for us that in terms of causing disease, particularly in terms of causing malignant mesothelioma, that the most, I think the word you used was poisonous fiber, is crocidolite, correct?

A. Yeah. The most potent toxin.

Q. And the least potent is chrysotile, correct?

A. Correct.

Q. So if you take the same exposure to crocidolite that you have to chrysotile, the crocidolite represents a much higher risk for mesothelioma, correct?

A. Um, it depends on how you determine the content, the crocidolite content and the amphibole content. If you use fiber burden analysis, the finding of a smaller concentration of crocidolite as compared to amphibole does not support that. It only states that after the event, amphiboles were more efficiently retained in the lung; whereas chrysotile escapes from that area, as demonstrated by the translocation

studies and other studies, concluding the fact that you can have tumor outside of the lung.

Q. I'm not sure how we got on to tissue digestion and fiber burden analysis. My question was simply: If you have the same exposure, the same level of exposure to crocidolite fibers versus chrysotile fibers, that same level of exposure is much more risky, carries a greater risk of mesothelioma if you are talking about crocidolite rather than chrysotile, right?

A. I had to explain to you what is the basis for that. What the basis is in the measuring concentration and comparing concentration. And I pointed out the fallacy of that approach. What is important is to know what was the concentration of chrysotile when the patient inhaled into the lung. So at the front end, not how many fibers are left at the back end.

Q. So do you have an estimate as to the relative potency? Because I've seen a variety of estimates of potency between crocidolite and chrysotile in the literature. Do you have one that you use?

A. Yes. My own is less than the one by and Hobson. They use 500 to 800 to 1. In my experience with the new data, if you were to calculate that, would be more like 50, 10 and 1.

Q. So --

A. I take it back. 100, 50 and 1.

Q. So the crocidolite in that -- in your range of carcinogenicity, the crocidolite is 100 times more potent?

A. Correct.

Q. There have been other estimates over the years that it went as high as 1,000 times more potent?

A. Yes. That's why it's a --

Q. Sure.

A. -- denominations in flux. It's pointing out that it's impossible to measure everything to the utmost degree, like you are trying to imply.

Q. So it remains important, then, when you are talking about exposures, if one asbestos fiber type is at least 100 times more potent than another in causing disease, it would be important to know the fiber type involved in the lower level exposures, right?

A. I didn't understand the question.

Q. Okay. Maybe I -- maybe I'm not being clear.

If crocidolite is 100 times more potent than chrysotile in causing meso -- are you with me?

A. Yes.

Q. And you are trying to evaluate a low level exposure?

A. Yes.

Q. It is obviously a more serious exposure if that low level is to crocidolite than if it is to chrysotile, right?

A. Yes. Except for one reason. Measuring an air sample

really impairs the identification of the fiber. The fiber is more precisely identified when it's lying on a filter and you can examine it by electromicroscopy and by spectography.

Q. Dail and Hammar -- Dail and Hammar. It's a book that you cited to several times and that you were cross-examined out of several times. It has a section beginning at page 581 that is titled much like some of the discussions that we've had today. "Is a threshold or minimal level of asbestos exposure or inhalation" -- it's got a slash -- "required for mesothelioma induction." Are you with me?

A. Yes.

Q. And it starts off with the proposition that you discussed with plaintiffs' counsel this morning, that is, "No minimum threshold dose of inhaled asbestos has been delineated below which there is no increase in the risk of mesothelioma." Correct?

A. Correct. But I'm not seeing where you are reading. Is that on the left column or the right?

Q. I was reading the first words in the paragraph.

A. Okay.

Q. Do I need to sort of maybe --

A. I can see it now.

Q. -- focus a little bit?

A. I can see it now.

A. I'm looking at it. I just couldn't place it because the

type is very small. I can see it now, yes.

Q. All right.

A. And I can see that there are numerous references, at least a dozen or more, to the same effect.

Q. Actually, there is six, but that is okay. The -- the concept that we just discussed about the difference in potency for fiber types, which may range from 100 times more potent to 1,000 times more potent, depending on whose science you are using?

A. According to your representation, yes.

Q. Then when you are talking about no minimum level that has been delineated below which there is no increase in risk, that is not saying that there is no level below which there is no cause. In fact, let's go through in that same -- bear with me for a moment, I'm trying not to read everything. Bottom of the page on the right-hand side. Let me make sure that I've got this so it's nice and -- maybe I can zoom in a little bit. All right. It talks about estimating a cumulative exposure of one fiber per milliliter year, one fiber year of exposure, which yields 650 mesos per 100,000, between 250 and 1,500 for crocidolite, down to about five for chrysotile, between one and 20, but as soon as you get down to the next level after one fiber year, when you get down one decimal point for a cumulative exposure of 0.1 fiber milliliter years, that is .1 fiber years of exposure, while

you still get 100 deaths from crocidolite, the risk for chrysotile level of exposure was probably insignificant.

So when you are dealing with low level exposures in the studies that you cite and to which you point, while asbestos in general has no known level at which the risk is not significant, within those same tests we find that as long as we distinguish fiber type, there are levels which they say the exposure is insignificant and the risk is insignificant.

So let me ask you this: Would it not help you to know the potential exposure level attributable to Mr. Haskins' use of gaskets in the Navy associated with Buffalo pumps? Wouldn't it help to know whether or not it hits a level that Dail and Hammar call insignificant?

A. That is the type of question I call around the world in 80 days. You mix a lot of concepts in there. The important one is that regardless of how you interpret it, or misinterpret what you are reading, the conclusion of the author is that there is no safe level below which asbestos is not the cause of cancer. That conclusion has been examined and reexamined by the scientists of the world, by groups of scientists that did it together, by regulatory agencies that try to determine a safe level, and by scientific organizations that took into account literature. And it is an accepted fact that below a certain level, you cannot rule out the possibility that it does cause it, primarily because

not every individual is alike. And these studies, particularly the epidemiological studies, rely upon means and affirmations. For you to try to say that it is an accepted concept by the scientific community that the no threshold applies only to chrysotile is to try to give crocidolite or amosite a magical power that one of their fibers can do it and millions of fibers of chrysotile cannot do it.

Q. I would take issue with the very first part of that long and rambling response in which you told me that I was misinterpreting the article. I don't think I did. But let's move on.

The -- when we talk about fiber years, one fiber year is an exposure of one fiber per cc eight hours a day for each working day of the year, which works out to about 2,000 hours at one fiber per cc over the course of a year. That is one fiber year, correct?

A. Correct.

Q. So if you get 25 fiber years, that's one fiber per cc for 25 years, right?

A. Yes.

Q. If you look at the exposure in Mr. Haskins case -- now let's just confirm again, you didn't read his deposition, did you?

A. Um, I don't believe I read it until later.

Q. You didn't read his deposition. You didn't read anything

other than the summary that his attorneys prepared for you, correct?

A. No. I obtain my information about exposure from several sources, from conversations with the plaintiffs' attorneys, with --

Q. I was coming to those. I said you didn't read anything.

A. With summaries that were provided to me.

Q. Right.

A. With whatever depositions there was in the case, and in many instances I rely upon reports or depositions of experts from the material sciences occupation medicine and industrial hygienists. So I take into account a totality of the information.

Q. So when you prepared your report in this case, you had read depositions of experts?

A. You can read the report and see what I said that I reviewed.

Q. Can't I simply ask you?

A. Well, I don't --

Q. Do you know?

A. I don't believe I saw that when I wrote the report.

Q. Exactly. So when you wrote your report in this case, the only information that you had about exposure to Buffalo pumps in Mr. Haskins case was a summary prepared by his attorneys, right?

A. And from conversations verbally that I had over the telephone.

Q. All right. And things that they told you that they didn't write down. I understand.

So in your report you state that while the ship was underway, you say once the overhaul was complete and the ship set sail, Mr. Haskins operated, repaired and maintained the engine room, machinery and equipment, including turbines, condensers, evaporators, pumps and valves. So while the ship was at sea, he was maintaining and repairing pumps, is that -- okay. So let's make sure that we know that. So he's repairing and maintaining pumps while the ship is at sea. You state that he went -- that when he was aboard the *USS Cony*, the ship underwent a major overhaul, and Mr. Haskins worked alongside the shipyard mechanics, and that mechanic from the Charleston Navy shipyard carried out the overhaul, removed and installed asbestos-containing materials. Is it your understanding that Mr. Haskins had exposure to asbestos-containing gaskets during the three months of the overhaul or during the entire time that he was on?

A. My understanding of it is that he worked primarily with gaskets and pumps, but as I say to my report, he was also exposed in a variety of ways, not only by being next to individuals doing the overhaul, but also at sea when he was

working near maintenance individuals. And the fact that these exposures are documented, I enter in my conclusion that all exposures, including the ones not related to gaskets, were part of the cumulative dose.

Q. All right. In fact, when you prepared your report and gave your deposition in this case, I believe you testified that you assumed that the overhaul lasted, quote, almost a year. Right?

A. I don't recall that.

Q. Oh, perhaps I can help you out.

A. The exact facts can be verified by looking at the record.

Q. My mistake. I'm looking at Cheshire. I need to look at Haskins.

Doctor, do you have a copy of your deposition up there? Otherwise, I can hand you this one.

A. I don't have a copy.

Q. Do you recall being asked at page 12 on -- at line 12 on page 26, "Do you have any information about how long the overhaul lasted?" And your answer was?

A. Let me look at it.

Q. "It lasted several months, almost a year, I think." Beginning at page 12.

A. The emphasis is on "I think".

Q. Yes.

A. Okay. What --

Q. Just make sure --

A. At that point factually is immaterial. The fact that I may have confused six months for a year is immaterial.

Q. Okay.

A. It is a long several months. My recollection is that it was a year, but if you want to verify, you can look up the record.

Q. All right. Well, let's assume, just for the sake of argument, that this wasn't a year, that this was, in fact, the three months that Mr. Haskins described in his -- in his deposition. And that let's assume that he said that he worked on ten or 15 pumps, ten or 15 pumps?

A. Okay. But are you talking about as part of the overhaul --

Q. Yes.

A. -- or his regular duties?

Q. Yes. Let's leave out his regular duties for a moment, because I think that perhaps you will find that he did not work on pumps during that time.

A. Well, I assumed that he did.

Q. You assumed that he did?

A. I receive information, I review information, and I form my understanding, and I assumed it was correct. If you want to present me different facts or give different hypothetical, I will consider.

Q. I was in the middle of doing that when I said let's assume. So let me start again.

Let's assume that he said that he worked on ten or 15 pumps. So let's take the side of that and say 15. Let's assume that he worked on gaskets associated with those pumps. Now, irrespective of the hypothetical that you were asked before which referred to a bonnet gasket on a pump, which is an item which does not exist, since there is no bonnet on a pump, let's assume that we are talking about flange gaskets, so there are two per pump. You understand?

A. Yeah.

Q. An inlet flange and an outlet flange, each of which has a flange face and a pipe flange face, a pump face and a pipe face, in between there is a gasket. Are you with me?

A. Yes.

Q. And let's assume, then, that for the 15 pumps, which is the high end of his estimate, we have therefore two pumps, two gaskets per pump. So 30 gaskets, right?

A. Right.

Q. And let's say he estimates that it took him at one point he says it took a half an hour to remove a gasket, and the other time he says it took an hour. Let's take the high end. Let's take an hour. If you've got 30 gaskets at an hour a gasket, you have 30 hours, correct?

A. Correct.

Q. And if you then divide that by the number of working hours in a year, which is 2,000 -- are you with me?

A. Yeah, I'm with you, but I don't need to be.

Q. Okay. Then you've got 30 divided by 2,000 yields .015, correct?

A. Correct.

Q. Multiply that by the midrange gasket exposure level, the average exposure level, even including the vast outlier of the Longo study, which gives you 2.79 fibers per cc, if you multiply .015 by 2.79, you get 0.04185 fibers per cc years.

Now, what I'm going to ask you is do you know whether 0.04185 fibers cc years of chrysotile exposure from these gaskets is sufficient to cause mesothelioma in this man with no further exposure, no other exposure?

A. As I explained to you, I don't give emphasis or rely upon quantitative measurements for several reasons: I'm not an industrial hygienist. I'm not a materials person. I'm not an occupational physician that works in those numbers. What I go by is the approach that is described here, my methodology. And I am backed up by scientific organizations, regulatory organizations and a number of respective investigators, including Dail and Hammar, that you don't need to know the cumulative dose, you just need to know that different exposures contribute to the total cumulative exposure.

So all your brilliant calculations do not enlighten me to answer that question.

Q. I appreciate my math being brilliant. I don't know that it is, it's just multiplication, but I'm delighted to be brilliant in math, something I have never been. I have never been brilliant in math before today. I'm pleased to be.

All right. When we are looking at your belief that in this case the exposure to -- well, scratch that. Let me back up just for one moment.

Let's assume for a moment that there is such a thing as a bonnet gasket on a pump, there -- there isn't, there are no bonnets on pumps, unless someone tied a hat on it, but it's a valve or -- a bonnet gasket is a valve word. Let's assume that there is a gasket on a pump and instead of talking about flange gaskets, we are talking about this bonnet gasket.

A. Between the body of the pump and the bonnet.

Q. If there were a bonnet. Then that cuts our number of gaskets in half because then instead of having two flange gaskets, we've got a single bonnet gasket. And that means that we are down to 15, which makes our calculations go down by 50 percent, correct?

A. Right. You are still in the area that I told you that is irrelevant for my determinations.

Q. Right. Because in order to determine whether it was a

cause, you don't need to know the quantification?

A. Exactly. I don't need to know where in the product the gasket is or is not.

Q. Right. When you are talking about risk in the asbestos context and there is no known safe level of asbestos in terms of increasing one's risk, that is not the same as saying that any level of asbestos is causative of disease. That is not the same thing, is it?

A. I don't know. Depends on what you are implying. What I'm saying is there is no safe level, meaning that there is no level that does not carry a risk of mesothelioma. And by the same token, there is no way for you to determine that value for an individual because of individual susceptibility.

Q. I fear that I may have gotten lost in that again. Let me try again.

Risk is not the same thing as cause, is it?

A. No.

Q. And you can have a tremendously heightened risk and yet have no disease that is caused, correct?

A. Correct. But if you have the disease, it's 100 percent, it may be the risk leads to only 50 percent of the cancer, but the individual, that is immaterial because they developed cancer.

Q. So let's put it this way: You gave us a series of potential causes of malignant mesothelioma. You said

asbestos, erionite, the sort of long-standing lung infection empyema, thorotrast, an injectable contrast drug used to highlight body features on x-ray, therapeutic x-rays, the old kind where they used to just blast you with x-rays, and idiopathical, which is to say we don't know what causes it, it's just genetic mutations that happen, right?

A. Right.

Q. So we've got six different ways for those cellular level, molecular level changes to happen, right?

A. Correct.

Q. And let's presume, just for a moment, that someone got mesothelioma because the cells in his body were transformed by one of the nonasbestos causes.

A. You are not talking about Mr. Cheshire anymore, correct?

Q. I'm not talking about anybody. This is hypothetical.

A. Okay. Right.

Q. If someone had a thorotrast-caused mesothelioma and it is 100 percent attributable to his thorotrast, are you with me?

A. Yeah.

Q. Even though prior asbestos exposures might have increased his risk, they did not ever cause his meso, correct? Not under our hypothetical?

A. No. In a patient, the thorotrast is another idiopathic agent. You cannot ignore the asbestos exposure.

Q. I am giving you a hypothetical in which we know as a

matter of scientific certainty.

A. Well, let me go back and say the operative communication that you did with me was when you did like that. Meaning there are less than five cases of any of those as compared to 3,000 cases a year of asbestos-related thorotrast is a few case descriptions, empyema is even rarer, and radiation is extremely unusual. So you cannot put in the same category an agent that is in the environment and in the working area of patients at the same par with thorotrast, the other ones which are only sporadic.

Q. I think you are missing the point. I hope so. I'm trying to get at a distinction between risk and cause. If you know that one thing and one thing only caused a mesothelioma, there are no other causes if you know that only one thing caused it, right?

A. Yes. But thorotrast is not the only one that can cause mesothelioma.

Q. I'm not saying that. I had no intention of saying that thorotrast is the only thing that causes meso. Listen carefully.

If you know what causes a mesothelioma, other potential risks are irrelevant once he's got it, right?

A. No.

Q. Oh?

A. You have to consider in your differential -- that is why

I took the trouble to verify whether or not he took thorotrast, whether or not he had radiation, whether or not he had a chronic infection -- you have to consider all the possible culprits in a thorough examination to arrive at a conclusion beyond a reasonable scientific doubt.

Q. Well, I appreciate the idea of a thorough examination, and I appreciate the idea of reasonable scientific doubt, because I'm not sure that, without knowing this man's exposure you can achieve that. But I'm really still on my hypothetical about the distinction between risk and cause.

So Doctor, one more time. Let's make it a little easier. If you get malignant mesothelioma from one of these nonasbestos causes -- pick one, I don't care if you use thorotrast or not -- if you get mesothelioma from a nonasbestos cause, then asbestos fibers that you may have inhaled over your lifetime did not cause it, correct?

A. The thing is because you found thorotrast --

Q. It's a yes or no.

A. Thorotrast is one cause. You cannot rule out if the person had unknowingly exposure to asbestos. You are trying to say that something exists that links that mesothelioma to thorotrast. There is not. There is something that links it to asbestos exposure by the characteristics of the pleural plaque, the asbestosis and the tumor itself. Thorotrast does not have a signal to that disease.

Q. I love how our hypothetical has grown plaques without my knowledge. Let's return to what I was talking about. And that is there are exposures -- and we've already identified them -- there are background exposures, there are low level chrysotile exposures, there are exposures that do not cause any increased risk for mesothelioma, correct?

A. There is no safe level.

Q. Even background. So the background can cause it?

A. No, I didn't say that.

Q. That is a safe --

A. I'm saying that you cannot determine the background. It's not a reliable feature. And there are no studies of patients exposed to background where the majority consistently got mesothelioma.

Q. So does background cause meso or not?

A. Not to my knowledge.

Q. Okay. So there is a level at which asbestos doesn't cause meso. It's called the background level, right?

A. No, it's not.

Q. Oh, okay.

A. Because --

Q. It does cause it?

A. Because the concept of background is fraught with difficulties to determine. And the reason it occurs that way is because someone may think that they were exposed only to

the background, but you must rule out other exposures, and that may be impossible. The patient may forget, may not remember, may not be aware of that happening. He may be living in a building that asbestos fibers are circulating in the air conditioning system.

Q. Like the man with the bag of asbestos in his basement. All right, sir.

So if I understand correctly, you will not exclude any source of asbestos, no matter how small, how minuscule from causing a mesothelioma in Mr. Haskins case, correct?

A. No. What I'm saying is there is no safe level that you can establish beyond a reasonable doubt that that level does not cause mesothelioma because each person carries with them a genetic makeup responsible for the robustness or not of its defense mechanism.

Q. Even though you admit that background levels don't cause mesothelioma -- in fact I think you said they don't cause disease at all -- even though you say that, you are unwilling to tell me that they are a safe level. That is fine.

A. No. What I'm saying is that there is no documentation of that and there is no data to support that hypothesis.

Q. There is no data to support the negative?

A. Yeah. There is no study where --

Q. Where they prove the negative?

A. They have good control of the environment at all times of

the day and under all circumstances.

Q. All right, sir.

THE COURT: Are you close to a stopping point? It's been two hours.

MR. MERIWETHER: Let's take a break. I feel that the equine is being flogged despite its death, and so --

THE COURT: Okay. All right. So we'll take a break. We'll start again in about 15 minutes.

MR. MERIWETHER: Okay.

THE COURT: Thank you.

(Thereupon, there was a brief recess.)

THE COURT: Okay, Mr. Meriwether, you are up.

MR. MERIWETHER: Thank you, Your Honor.

BY MR. MERIWETHER:

Q. Dr. Bedrossian, picking back up for a moment, I believe you've testified already that you used the term cumulative exposure to establish causation, correct?

A. Correct.

Q. When you are looking at cumulative exposure, you exclude or include background levels?

A. Background level exists, but by itself is not a cause of mesothelioma.

Q. Thank you. When you look at exposures, the cumulative exposure, you refer to something called occupational exposures, correct?

A. Correct.

Q. And in fact, you were queried about that in the fall of last year in the companion case of Hinsley. Let me put on the Elmo something that might help you remember. "If someone has occupational exposure, in your opinion" -- and I'm beginning on line 20, you can probably read it -- "If someone has occupational exposure, in your opinion do you need to know the frequency, the regularity and the proximity before you will say that that occupational exposure was a significant exposure?" And the answer is, "You need to establish that it happened, you don't need to quantitate it."

A. Right. Quantitated does not refer to proximity, intensity and regularity. You need to have, as part of the definition of a significant exposure, to have those three characteristics.

Q. And so where on the next page at -- on page 42, where you say that when Mr. Hinsley for three years was in a ship environment and was a machinist, that it is assumed that that was of long duration, that he did work in proximity to the asbestos because he actually handled the asbestos insulation, and that there was enough frequency because he did that routinely on a daily basis, now, so when you have an occupational exposure, are you entitled to assume that there is regularity, frequency and proximity?

A. No. I was describing that situation. He was in a ship,

he was there for eight hours.
Q. So that gentleman worked for three years and you say that he had daily exposure?
A. I can't remember the facts of the deposition.
Q. That's fine. Let's skip over a bit. How do you define an occupational exposure?
A. Um, the exposure that occurs while the individual is working with an asbestos-containing product that releases fibers that become visible in his work area.
Q. That's occupational?
A. Right.
Q. Okay.
A. As opposed to para-occupational in changing brakes in his backyard, for example.
Q. All occupational exposures contribute to the cause of a man's mesothelioma?
A. Define what you mean by "all occupational exposures".
Q. Every one, excluding none.
A. Well, are you talking about insulators, as opposed to joint compound users, or are you talking about every exposure in the world that occurs when someone is doing a job?
Q. All right, sir. I began by asking you to define what you think is an occupational exposure. I'll ask you that again. What is an occupational exposure?
A. I already answered, but I'll do it again.

Q. That's fine. You can stick by your answer.
A. When the person is occupied in doing a work activity.
Q. So now I'll ask you the second part. Using your definition of an occupational exposure, is every occupational exposure causative, does it contribute to the cause of a man's mesothelioma?
A. No. That exposure deserves to be called occupational, but it does not mean that every occupational exposure is equal or the same.
Q. Whether it's equal or the same is not the issue. What I'm asking is whether or not it contributes to the cause of a man's mesothelioma.
A. Yeah. If it is an occupational exposure, I understand -- my understanding that I form is that it is close to the source of the asbestos, frequent and repeated.
Q. So that takes us back to page 42 of what we were looking at a moment ago, where you answer at line 14, "Yes, occupational by definition implies that there is sufficient proximity, dose and frequency as opposed to background exposure." Right?
A. Correct. It has the characteristics that are important: dose, frequency and proximity.
Q. So those are simply assumptions that you can reach because it's an occupational exposure?
A. No. These are features that define the exposure as

occupational.

Q. Let's talk about some of the things that you briefly went through with plaintiffs' counsel this morning. A number of different -- number of different slides that were shown. Here is one that supports the idea that there is no safe threshold and single-day exposures may be potentially causative. Of the items on this page, of the different groups or organizations on this page, only the EPA, the NTP and IARC were cited in your report. The rest -- were the rest suggestions by the attorneys or did you come up with them after the fact?

A. No. There was no suggestions from the attorneys in my report. By the --

Q. I think you missed the point.

A. By the limitation of the report, I did not list all my sources.

Q. I see. But they weren't listed in your report. I noticed that the next page which has the Thoracic Society with a fallout and a paper. That wasn't listed in your report, was it?

A. Right. I did not pretend that my report included every piece of evidence that answer my understanding and conclusions. For example, for those -- each of those consensus papers, they considered hundreds of articles leading them to form that understanding. Just here I can

remember papers by Wiggins, Hodgson and Markowitz, many others that come from different disciplines, including epidemiology or work studies, or co-work studies.

Q. Let's take a look at another one, Mr. Levin's article, Jan Levin's article, not listed in your report, right?

A. No, it's not. If you can read my report, you would know the answer to that question.

Q. All right. Next, Morris, Greenberg and Davies, not in your report, correct?

A. No.

Q. No, not correct or no, correct?

A. No, it was not listed.

Q. I thought so. Rodelsperger, in your report or not?

A. No, not in my report.

Q. Not. Lacourt, in your report or not?

A. Well, I have to look at it.

Q. I will represent to you that it was not.

MR. FINCH: Objection. It is in his gasket bibliography.

MR. MERIWETHER: In the gasket bibliography. Is that part of his report?

MR. SWETT: It's marked as an exhibit in his deposition.

MR. MERIWETHER: I'm asking if it was in the report.

THE WITNESS: In the report you can read it for yourself. Why do you need to ask me?

Q. Because I would like to make a record for the purposes of this proceeding, if that's okay.

I'll try to be brief. The ATS Society 2003 statement in your report?

A. No, it's not.

Q. Okay. I think that is enough. I have a number, but I'll move along.

The methodology that you use to attribute asbestos exposure, or to attribute a mesothelioma to an asbestos exposure, if I understand correctly, is the determination that there is no safe level and a series of assumptions based on occupational exposures. Am I missing anything?

A. Yes.

Q. Is there more?

A. Yes. You are trying to simplify it. It is more complex. The methodology includes all the steps that I enumerated earlier. It starts with a medical consideration of the facts of the case, including the history, particularly the occupational history, the hobbies, where the patient lived, followed by looking for signs, physical signs of exposure, including x-rays, showing pleural plaque or asbestosis, considering the latency period, if that is consistent with the condition, followed by a differential diagnosis where I

consider alternative causes, physical, chemical and biological.

Let's say if I find that the patient had radiation, then I will probe further and I will see what part of the body was radiated or other details.

Q. All right.

A. The holistic approach.

Q. Doctor, you've done a grand job of explaining to me why you can attribute mesothelioma to asbestos exposure but not to attribute mesothelioma to a single exposure as a cause.

Let me ask you this --

A. Well, because I never do.

Q. You never do?

A. A single cause. I do it to because -- because being cumulative exposure.

Q. You used this chart and it was referred to as a dose response curve. In fact, that is a linear representation, not a curve, correct?

A. Yeah. But I don't think it was implied that this is a dose response. That is a comparison between different causes, whether they are more prone to develop mesothelioma or not.

Q. So when you get down to the bottom down here and it appears to indicate that only at zero exposure do you have zero disease, this part of the graph is not necessarily true.

It might be that it goes very low and then comes up, right? We just don't know.

A. This is a graphic representation of ideas. It is not specific to describe or illustrate numerical measurements.

Q. Do you have any dose response curve for low level exposures to asbestos?

A. I don't personally own them, but I have seen it in the literature.

Q. You've seen a dose response curve for background level exposures?

A. No, I don't remember that.

Q. I didn't think so. All right.

Likewise, the next page appears to have straight lines. These are not representations that are scientific, these are simply graphic communications of an idea, right?

A. Yes. It is different than the other one. This one implies that you are measuring, because it has a scale.

Q. Does imply that, yes. All right.

One of the things that you have cited to is the Helsinki Criteria. One of the defense doctors in this case, Dr. Roggli, was invited to the Helsinki Criteria; you were not, correct?

A. No, I was not.

Q. Dr. Roggli was also described earlier as somebody who testifies really for defendants in this litigation. You are

aware, since you remember Dr. Roggli as far back as his student days, you are aware in the beginning in the early days of the asbestos litigation he was testifying for plaintiffs, correct?

A. Yes. I remember that he testified for both sides.

Q. When we are talking about the Helsinki Criteria, it requires that a past exposure to asbestos be significant before you can attribute meso to asbestos in the absence of other markers, right?

A. What they say is that you have to have an occupational exposure. And in delving into that, they say an occupation history of brief or low level exposure should be considered sufficient for mesothelioma to be designated as occupationally related.

Q. And if you look at the curves or the noncurves, but the straight lines that were intended to indicate an idea which we looked at a moment ago, the shorter the duration, the higher the necessary dose, right?

A. Yes. That's correct.

Q. So you are going to have to have a very high dose if you have a very short duration?

A. Correct.

Q. And indeed, in that statement it is talking about asbestos generally. So a shorter duration with a fiber type of crocidolite could be 100, 500 or 1,000 times more potent

than the same exposure to chrysotile, right?

A. Well, I think there is summation. They were not addressing what you are describing. They are talking about in general. They are talking about averages and the construct of the concept that you represent in the graphic format.

Q. So Helsinki does not say that each and every substantial contributing -- excuse me -- Helsinki does not say that each and every exposure is a substantial contributing factor to causing mesothelioma, does it?

A. Yeah. Whether or not they say cumulative or not, they say that an exposure that is low cumulatively has to be considered sufficient for mesothelioma.

Q. Let's just answer the question and then move to the explanation.

Helsinki does not say that each and every exposure is a substantial contributing factor to causing meso, does it?

A. I don't recall now if they mention that.

Q. Say that or not?

A. Specifically. But if you read the paper and are interested in comprehending the scientific concept, that the conclusion that is reached --

Q. And in fact --

A. If you are trying to deny it or trying to find some way

out of the conclusion, it may be different.
Q. It does not say that cumulative exposure causes mesothelioma, either, does it?
A. I don't know if it does or not.
Q. But you are the one that cited it.
A. I'm sorry?
Q. You are the one that cited it.
A. No, I did not cite Helsinki that specific and --
Q. Would you like to withdraw your reference to it?
A. No. If I wanted to cite specific articles that were designed for that and list it, I would have chosen the ones that address only cumulative dose; whereas Helsinki is a comprehensive statement.
Q. All right. Let's turn -- all right, Doctor. Let me finish with this thought, I'll try to be brief: Dail and Hammar, which is the textbook that I believe you cited the most often this morning in your discussions, in it it simply states that for chrysotile asbestos exposure at a .1 fiber year dose --
A. Can you tell me where you are?
Q. Oh, sure. I'm right where I was before. This is the bottom of page 481 at the bullet point that begins, "As set forth in their review" -- and it's talking about Hodgson and Darnton. It gives a series of descending concentrations, starting with 1.0 fiber years, moving to 0.1 fiber years and

moving down to 0.01 fiber years for both .1 and .01 fiber years.

A. I see that.

Q. The risk of mesothelioma from chrysotile is, quote, "probably insignificant".

MR. FINCH: Objection. Under the rule of completeness, I would ask that he read the rest of that sentence.

THE COURT: Go ahead.

Q. "With a highest arguable estimate for four deaths per 100,000." Period is the rest. So Doctor, once again, we wish to apply that science to the Haskins case. Do you have any idea what the fiber year exposure to gaskets associated with Buffalo pumps would be?

A. No, because I don't use that numerical figure as a deciding yay or nay factor.

Q. Understood. Thank you.

MR. FUSCO: Your Honor, if I can have one second to move my stuff up?

THE COURT: Sure.

MR. FUSCO: Your Honor, David Fusco on behalf of Crane Co in the Cheshire case.

Before getting started, I would like to expedite things. I think it goes without saying, but if possible I would like to incorporate Mr. Meriwether's arguments in the

Haskins case so I can minimize what I need to go into in my examination.

THE COURT: Thank you.

CROSS-EXAMINATION

BY MR. FUSCO:

Q. Good afternoon Dr. Bedrossian.

A. Good afternoon.

Q. I'm going to try streamline things a bit and not cover things that have already been covered, at least not anymore than necessary, so I'll be jumping around a bit. So bear with me.

First thing I would like to address with you is obviously we are here pursuant to a motion related to your testimony. And I would like to read from you -- read for you a passage from plaintiffs' opposition to defendants' motion to see if you agree with the statement that plaintiffs present. At the top of the page states, "With regard to causation of mesothelioma, therefore, the issue is not which occupational exposures to asbestos can be considered to substantially contribute to the mesothelioma risk; but rather, whether any such occupational exposure can be excluded as not proven to contribute."

Do you agree with that statement?

A. Well, a little bit convoluted but the meaning of it, unless you are inclined to prejudge what it means, what they

are saying is that it is cumulative exposure, and within the cumulative exposure, you have some that you don't need to say without that it doesn't cause mesothelioma. They are saying that all exposures add up to a cumulative dose.

Q. And to clarify and keep things moving, Dr. Bedrossian, do you agree with the statement as plaintiffs presented it in their brief there on the screen?

A. Yeah, I agree with it, with the explanation that I gave you.

Q. Do you agree with the statement that is written in the paper, not the explanation you gave me, I just want to know, do you agree with this statement as it's written? And if not, please explain why you disagree with what plaintiffs stated in their brief.

A. Um, I agree with it.

Q. All right. Thank you.

Now, you mentioned several times throughout your testimony today, Dr. Bedrossian, that you do not believe there is a safe level of exposure to asbestos, correct?

A. Correct.

Q. And I believe at one point in your testimony you stated in regard to some of these government agencies and health organizations that as it related to OSHA being the most important one, you said OSHA PEL is the important one for purposes of regulation. This is what OSHA had proposed. And

you said that OSHA's statements were the most pertinent, I believe; is that correct?

A. Having to do with regulation, yes. That is the agency charged with doing regulation. It does not mean that it is the most important of all the papers that I represented in the slide.

Q. And is it your position that OSHA has never taken the position that certain level of exposures approaching the permissible exposure limits are safe and acceptable?

A. That's correct.

Q. And I think you have seen statements like this or documents like this already in today's examination from the United States Department of Labor website Occupational Safety and Health Administration statements of OSHA. I would like you to --

A. Can you increase your magnification so I can read it?

Q. I will do so. And do you see there that OSHA has stated, this is monitoring asbestos levels in the air following the World Trade Center, that, "Asbestos levels remain safe and consistent ranging from nondetected to .086 fibers per cc." Do you see that, that OSHA statement?

A. I see it saying that.

Q. Thank you. And we talked a little bit earlier about some of your reliance materials, Mr. Meriwether did, and your gasket bibliography. And we established that was disclosed

at your deposition but not cited in your report, correct?

A. Correct.

Q. And one of the articles cited in your gasket bibliography is an article entitled, "Exposure to airborne asbestos during removal and installation of gaskets and packings, a review of published and unpublished studies." The lead author was Amy Madl; is that correct?

A. Yes, that is it. One by stand-alone institute and deviates from the general understanding.

Q. It's from -- did you say it's from a stand-alone institute and deviates from the general understanding?

A. Correct.

Q. Okay.

A. From at least my understanding of the entire literature.

Q. Taking a look at this, the abstract here, you note that the Madl article, to refresh your recollection, is a metananalysis. They didn't review any exposure levels, they reviewed other people's studies and did an analysis of, I believe, 11 published and unpublished studies?

A. Yes. And that is the reason why it is subject to interpretation. Because in doing a metananalysis, the authors have an opportunity to select which articles they are going to give more weight and which ones they are going to give less weight.

Q. And you know -- we'll come back. Let's jump ahead and

look real quick at the articles that they did include. Moulett and Malick, right there, that was one of the gasket studies you talked about earlier and relied on in your direct examination, correct?

A. Correct.

Q. And here is Longo, Egeland and Hatfield, 2002. They included that study as -- well, that is another one you relied on?

A. Correct.

Q. How about Fowler? That one is in your gasket bibliography?

A. I can't see it here.

Q. Oh, I'm sorry. I'm looking at the document, not the screen. Fowler, 2000. It was the -- I believe this is the ban --

A. Yes, I can see that.

Q. Okay. And Boelter. That's another one that you have listed on your gasket bibliography, correct?

A. Yes.

Q. They included each of those studies in their analysis, right?

A. Right.

Q. And Longo, in fact, is the one that reflects the highest reported numbers in the literature as it relates to gaskets and packing, correct?

A. Correct. But let me make it clear that there is no special significance for the reference appearing in my bibliography. I did not select my bibliography based on the results. I submitted this many as examples of articles. There are many articles that are significant and may not be represented in here.

Q. Okay. Since this is our opportunity to examine the foundation for your testimony, I want to clarify, we didn't -- you didn't cite any gasket and packing-related materials in your report, any gasket and packing literature studies for your opinions in your report, correct? They were disclosed in your bibliography?

A. Correct. And I explained to you why.

Q. And Dr. Bedrossian, are there any studies that you rely on that are not listed in your gasket and packing bibliography for your opinions as it relates to gasket and packing exposures?

A. Yes. I have an extended bibliography which I prepared for today's hearing.

Q. Have you given that bibliography to anyone? I haven't seen it. I'm wondering if anyone has seen --

A. I did it within the last few days.

Q. Well, we'll leave that for another day, then, sir.

Going back to the Amy Madl article. I would like to turn your attention here to the conclusion here. And to keep

things moving, admittedly the article states, "In all but one of the studies relating to the replacements of gaskets and packing, all but one" -- she's referring to Longo -- "in all but one using hand-held tools, the short-term average exposures were less than the current 30-minute OSHA excursion limit of one fiber per cubic centimeter, and all of the long-term average exposures were less than the current eight-hour permissible exposure limit, time weighted average of .1 fiber per cc."

That is what they found when they averaged the numbers from those studies that other people performed, correct?

A. Right. Besides the fact that being below PEL is meaningless because of individual susceptibility. This exemplifies the fallacy of someone who is not a trained scientist or has knowledge of biology to interpret data.

Q. OSHA doesn't think that is meaningless. .1 fiber per cc, we are looking at .086, that is 86 percent of the PEL. The Federal Government, protecting citizens, is stating that those levels are safe and consistent. So OSHA thinks there is meaning in that number?

A. They don't say it's safe.

Q. On the screen there, Dr. Bedrossian, "Asbestos levels remain safe and consistent ranging from nondetected to 0.086 fiber per cc." That is what the document states.

A. That was applicable to that circumstance. There are many more NIOSH statements, many more EPA statements, many more OSHA statements.

Q. Applicable to that circumstance?

A. And they all have been considered by scientific bodies and by regulatory agencies where the Government contradicts in the language or someone may try to take this out of context and say that this here trumps everything else that has been published.

Q. I'm not talking about any of those studies. I'm talking about the OSHA PEL and time talking about the numbers reported for gaskets and packing averaged below the .1 PEL, a number that OSHA -- that it would be the PEL, 86 percent of the PEL, these were measurements measuring downtown New York City that OSHA is releasing in a press release to the public to inform them that these numbers and these exposures are safe and consistent. You disagree with the position of OSHA?

A. Yeah. With the way it is and the way it tries to portray it in a context that I don't know if it belongs, yes, I disagree with that. Because there are many more statements that they have made and those statements, plus the entire body of literature, universal scientific literature, led to conclusions that needed the participation of multiple experts, many more than just someone doing a press release.

Q. Not someone doing a press release, the Occupational

Safety and Health Administration of the United States, correct?

A. Well, you know, I don't know the exact branch they belong to.

Q. And the one study that was relied on in the model article that had the -- you mentioned had the highest numbers related to gaskets and packing, that was Dr. Longo, correct?

A. Correct.

Q. And Dr. Longo is affiliated with a group called Material Analytical Services, also known as MAS, correct?

A. Correct.

Q. They have done a series of work practice studies regarding gaskets and steam systems, brakes, other products and various things?

A. That's correct.

Q. And in his work practice studies, I believe there are a couple on your gasket bibliography, there are a couple of references to Dr. Longo. Am I correct you are relying on Dr. Longo's work practice studies to support your opinions in the Cheshire case?

A. They are part of the totality of information that I considered.

Q. And would it affect your reliance on those materials if Dr. Longo has stated under oath that, "Without an opportunity for my firm to perform a case-by-case evaluation of the

alleged exposure, I believe it is not proper for other persons not affiliated with MAS to select workplace simulations and attempt to use those simulations in a case." Would it surprise you if he said that?

A. Continue. Read that again.

Q. I'll show it to you. We are looking here at, this is Superior Court, State of Washington for Pierce County, Dr. Longo, you will see, signed it back here about a week ago. And you will see here, as a material scientist, he describes that he's tested products, and he talks about different types of gaskets he's analyzed. And this is Dr. Longo taking the position when another expert was trying to rely on his studies that that would be inappropriate. Do you see that there?

A. Well, I don't follow his conclusion. What he's saying is without an opportunity of him seeing the individual observations in the evaluation, he cannot deem this or bless it as the proper conclusion if somebody not affiliated with his institute, meaning not using the same technique, not using the same quality assurance; and therefore, the two simulations are not comparable. If it would be comparable, if they use the exact same technique, or even better, if the person doing the measurements was the same or trained together, because these results are -- have subjective aspects to it.

Q. Fair enough. Did you do anything in this case to compare whether Mr. Cheshire's alleged exposures from work with gaskets and packing -- or I'm sorry -- with gaskets on Crane Co valves were directly comparable to the circumstances and all the details you just described in any of Dr. Longo's studies?

A. No, I did not. I remind you that was the totality of the evidence that I saw that led to my conclusion, not a specific portion.

Q. Now, you testified in your deposition, I quote, you don't "have any opinion or conclusion regarding any specific product or any specific product manufacturer in the Cheshire case, correct?

A. Yeah. I don't have a conclusion that is specific and limited to that product.

Q. And, in fact, when you were hired by Waters and Kraus, they sent you -- in the Cheshire case, they sent you a letter about your retention, and you were asked about that earlier. And I think it was described that you were asked what -- whether he had mesothelioma and what the cause of that mesothelioma was. Do you recall that?

A. Yes.

Q. I would like to show you the letter that they sent you -- and to clarify for the record here, the questions they asked were actually whether Mr. Cheshire has mesothelioma and

whether his mesothelioma was caused by exposure to asbestos, correct?

A. Yes, because they identified to me on the telephone that this was the exposure. The information I get from the attorneys is not restricted to a letter or to a summary of the work history, it includes also verbal communications.

Q. And then in addition to those verbal communications, it includes documents they gave you like the work history sheet, correct?

A. Correct.

Q. The work history sheet that said Mr. Cheshire wore respirators during a certain period of his career, you circled that. That is your circle?

A. No, I didn't circle.

Q. This was marked -- this was your copy marked as Exhibit 4 at your deposition.

A. I don't put circles in exhibits.

Q. We'll move on. I think it's not important for our purposes right now.

And their work history sheet listed all of these products: gaskets, valves, pumps, air conditioning compressors, boilers, feed tanks, distilling plants, evaporators, purifiers and various brands of those products under each category, correct?

A. Correct.

Q. It is your opinion that all of those exposures were a part of the cumulative exposure and that cumulative exposure is what caused Mr. Cheshire's mesothelioma, right?

A. In reading this, I don't conclude that because they list it -- they are saying that he was exposed to all of these.

Q. Which ones were they saying he was exposed to?

A. Well, the ones that was presented to me, and I understood to represent the exposure and to which I was giving an answer has to do with gaskets.

Q. Which exposures -- so the only exposures that the attorneys for Waters and Kraus told you about to reach your conclusions as to the cause of Mr. Cheshire's mesothelioma were gaskets. Is that what you said?

A. No. They asked about that connection. And as I said, in my holistic approach and my methodology, I considered alternatives that I could gather from patient history. Since he was in the Navy, I definitely considered thermo insulation in pipes and boilers and other parts of the ship.

Q. Is there any manufacturer products listed on the work history sheet that you do not think contributed to the cumulative of Mr. Cheshire's cumulative exposure?

A. I really don't have enough information to answer that.

Q. Well, let's go to the valves here. The Leslie, Valin and Crane. Do you believe -- do you have enough information to answer it about those three, which ones of those would have

contributed to the cumulative exposure and which ones didn't?
A. The ones that are contained on the facts of the case. The ones that have been identified by special scientists who do that type of work.
Q. Did you?
A. I don't address myself brand names.
Q. You don't know specifically whether any of those three, Crane, Leslie or Valin, specifically those brands, contributed to Mr. Cheshire's cumulative exposure, do you?
A. I don't have the facts to answer that question.

MR. FUSCO: Those are all the questions I have, Your Honor.

THE COURT: Okay. Thank you.

MR. GEORGE: I've got a little bit of redirect.

THE COURT: Sure.

REDIRECT EXAMINATION

BY MR. GEORGE:

Q. Let's start where we just left off. If Mr. Cheshire testified in his deposition that while he was in the Navy working on the repair ship in the valve shop, that he replaced bonnet gaskets, uncraned valves routinely during the period of time that he was there and that when he was on the *USS Henderson*, again, he did similar work where he is replacing asbestos insulation on valves would that serve as a basis for you to determine whether that exposure was

causative?

MR. FUSCO: Objection, Your Honor. Lack of foundation. The witness already testified as to what he was aware of, what he relied on in his report. We can't add facts into the record at a *Daubert* hearing to support his testimony.

THE COURT: Okay. Go ahead.

THE WITNESS: Present a hypothetical now, so I don't follow what the attorney said.

Q. In your deposition, you were asked prior to presenting your report whether you had read Mr. Cheshire's deposition, and you had, correct?

MR. FUSCO: Objection, foundation. Misstates the testimony of the deposition.

THE COURT: We are at a *Daubert* hearing. I'll mark your objection. Overruled.

Go ahead.

Q. Did you have the --

THE COURT: We are going to have it out and then I can just disregard it.

Q. Just for the purposes of closure on this, did you have the opportunity to read Mr. Cheshire's deposition before the report was sent to the defendants?

A. No.

Q. I want to ask you, this thing that was put up here about

Dr. Longo, he's talking about automotive gaskets and why you can't use industrial gasket materials in the workplace to determine automotive gaskets. Does this case involve automotive gaskets at all?

A. No.

Q. You were asked about Dr. Madl's study. And you understand that Dr. Madl's study was actually -- first of all on the bottom, "Although we initiated this evaluation, financial support for the underlying research was provided by a pump manufacturer involved in asbestos-related litigation regarding gaskets and packing." And that one or two of the authors has served or may serve as an expert witness in related litigation. You are aware of that?

A. Yes, I was aware. And that is what I meant by say stand-alone institutes without clear evidence of good practices, quality assurance, and therefore reliability.

Q. You were asked some questions about Hodgson and Darton. In your book, one of the things that you have is the recent 2014 WHO chrysotile asbestos?

A. Yes.

Q. And it should be -- I think it's the second to last tab. Now, I think in your -- in one of your responses you indicated that Hodgson and Darton changed their numbers based on additional data they received. Do you recall that?

A. That's correct.

Q. And, in fact, they -- in this monograph, they comment on the fact that -- if you turn to page -- turn to page 29 and 30. On page -- the end of 29, they say, "The study of textile workers in North Carolina was not included in their original metananalysis. Based on an approach used by Hodgson, the authors of the North Carolina study estimated deaths and then Hodgson had to go back into the literature and rephrase the 500 to 100 to 1 ratio."

A. Correct.

Q. And I think you testified that it was now 50 to 10 to 1?

A. Yes. That is what I gather from my understanding of the literature.

Q. Doctor, we look at what IARC said. They said that the working group noted that there was a high degree of uncertainty. It says that, "The IARC working group noted a high degree of uncertainty concerning the accuracy of the relative potency claims derived from Hodgson, Berman and Crump analyses because of the severe potential for exposure misclassification in these studies."

Can you tell us what that means when you do a metananalysis and there is misclassification? What does that mean?

A. Well, what it means is that they may interpret the term differently. It may also mean that the mold of quantitation is not reproducible, it's not accurate, and it doesn't occur

in every case. So this reflects the difficulty or the impossibility of you to relate or to reduce to a given number a complex scientific problem.

Q. And has the EPA or any government agency ever accepted the potency numbers that Berman and Crump or Hodgson and Darton proposed?

A. No, they have not. They have gone as far as to say chrysotile is less potent, but it is a cause of mesothelioma.

Q. And over the course of time in the literature, there has been many different researchers that have posited what the relative potencies may be, correct?

A. Yes.

Q. And some, like Dr. Nicholson, thought it was 1 to 1 to 1, others 2 to 1, 10 to 1?

A. Correct.

Q. And your best estimate, based on your review of the literature, is that chrysotile is less potent?

A. Yes, it is, but it's not innocuous.

Q. It's not --

A. Innocuous. It's not harmless.

Q. Got you. You said at the beginning that it's not necessary for you to know the exact amount of dust generated before you can reach an opinion about causation. Why is that?

A. Because number one methodology for measuring both in

suspended air and in tissue is really fraught with complications. No results are the same between one laboratory and the other, or within two different measurements in the same laboratory performed at different times.

Q. Now, you had mentioned earlier you were talking about the fact that the chrysotile fibers translocate into the body. And I think you were about to talk about the different sites that mesothelioma can occur. Are there any sites outside the lungs where chrysotile can translocate to and can cause mesothelioma?

A. You can go to the analysis of the -- to have mesothelioma, it requires for you to have mesothelial cells in that area. If the asbestos reaches an organ, for example the kidney where there are no mesothelial cells, the tumor would be a renal tumor. Mesothelioma is a tumor linked to cirrhosis, areas of the body where mesothelial cells exist.

Q. And so you can get a mesothelioma of your testicle sack, you can get a mesothelioma of your paracardial sack surrounding your lung, you can get it in your abdomen?

A. Correct. Because all of those sites have mesothelial cells.

Q. And what does that tell you about the ability of these chrysotile fibers to get out of the lung and throughout the body?

A. Well, Dr. Suzuki did a series of experiments or wrote different papers, and later on in the early 2000s, he demonstrated the presence of asbestos fibers in the urine, in the kidney, and on places, therefore showing that asbestos fibers travel throughout the body, first from the lung to the pleura, and then from there to anywhere they wish to go.

Q. And which of the different fiber types is the fiber most likely to translocate from the lung to somewhere else?

A. Well, chrysotile is the most likely to translocate from the lung to the pleura. Now, beyond that, I don't think there are studies where they use isolated fibers to decide if the amphiboles are any different.

Q. You were shown this in cross-examination. This is the study by Iwatsubo, "Dose response relation at low levels of asbestos exposure in our French population-based case control study." And you were shown what were actually citations to other articles on page 141. You were asked about this paragraph about, "Some indication of the effect of exposure that is low level by brevity of its duration comes from industrial cohort studies." They are pointing out other studies, like 47 was a study from Leclerc.

A. Correct.

Q. Somewhere in Wittenoom in Western Australia.

And then you have Selikoff is indicated for one of them, his study of insulators. What they are saying here is

that these studies don't seem to show any cases of mesothelioma for brief exposures, but they then say these cohorts do not provide data to allow us to examine the effect of the low intensity exposure. What does that mean?

A. It means that not every study is quantitative. So they have not measured the levels, or if they measure, it may be in a different place. So the studies vary according to the methodology used by the author.

Q. What these authors concluded based on the data that they had is they found, "A clear dose response relationship between cumulative exposure to asbestos and pleural mesothelioma in a population-based, case-controlled study with retrospective assessment of exposure, a significant excess of mesotheliomas was observed for levels of cumulative exposure that were probably far below the limits adopted in many industrial countries during the 1980s." What do they mean by that?

A. They mean that low levels in a susceptible individual because of his being who he is, meaning his genetic makeup, that event can occur. But there are many more articles than that. In a report, even in a consensus report, it is impossible to quote every article contributing to that conclusion. But in my handy examples here of Dr. Wiggins, a Dr. Hodgson, Dr. Markowitz, which the same conclusion. Doctor Markowitz is particularly important because it's from

2015. So it's more comprehensive set of observations.

Q. You were -- you were asked some questions about whether exposure to ambient air can cause disease. Is there a difference between whether a substance can cause disease or whether we can prove it causes disease?

A. Yes, there is.

Q. And when we look at ambient air, how would you ever be able to determine whether somebody's exposure to asbestos that is at the level of .00005 causes disease?

A. There is no way I can do that.

Q. Why is that?

A. Because there is no characteristic feature, either the term scientifically or by observation, that link to low exposure. There is no identification of the exposure.

Q. How big a population would we have to look at to determine whether there is an increased risk at that level of exposure?

A. Millions of people.

Q. And is there anybody that we can put in the control population that wasn't exposed to asbestos so we can see if there is a comparison?

A. It's impossible to come up with that.

Q. And so when the Helsinki Criteria, when they talk about these type of exposures, when we are talking about these low level, ambient exposures, they say that, "Mesothelioma can

occur in cases with low asbestos exposure; however, very low background environmental exposures carry only an extremely low risk." Are they saying that there is no risk?

A. No, they are not.

Q. And when we take into consideration individual susceptibility, are we able to say that there is no risk at these very low levels?

A. There is not.

Q. And in either one of these two cases, are we talking about a low level exposure when you have a man that is on a Naval ship for a period of months or years?

A. No. There are considerable exposures.

Q. And you were asked about some different citations and whether they are in your report. In your report you did cite to Dell and Hammer, correct?

A. Yes.

Q. And are you aware of whether Dell and Hammer has citations to the various articles that Mr. Meriwether points out?

A. Absolutely. Particularly in the new edition of the book more so than before.

MR. GEORGE: One second, Your Honor.

MR. FINCH: One point, Your Honor. I'll just --

MR. GEORGE: It's easier for him.

THE COURT: Sure. Go ahead.

BY MR. FINCH:

Q. You were asked questions by Mr. Meriwether where he did, I believe you called it, elegant mathematics about the -- in the Dale -- in the Dale and Hammar book. Do you recall those questions where they were talking about level of exposure, the risk was probably insignificant. Do you recall those series of questions for chrysotile?

A. Yes, I recall.

Q. Okay. And am I correct that what the authors are talking about, they are talking about the analysis done by Hodgson and Darton; is that correct?

A. Yes.

Q. Do the authors of the textbook, when they ultimately reach their conclusions that no lower minimum threshold level of exposure to asbestos has been delineated below which there is no risk, is there an exception for chrysotile in that statement?

A. They don't specify one or the other.

Q. And is there any exception for chrysotile on their conclusion that when there are multiple asbestos exposures, each contributes to cumulative exposure; and hence to the risks and causation of malignant mesothelioma was at appropriate latency interval. Was there any exception for chrysotile in that?

A. They don't make any difference. They do not address

that.

Q. And is this book one of the things that you rely on as part of your methodology in assessing causation in asbestos mesothelioma cases?

A. Yes, it is.

MR. FINCH: No more questions.

MR. MERIWETHER: Literally probably two questions on recross, Your Honor.

RECROSS-EXAMINATION

BY MR. MERIWETHER:

Q. Dr. Bedrossian, you were asked whether or not Dail and Hammar was just referring to Hodgson and Darton. In the second quote they are just referring to Peto, right?

A. Referring to whom?

Q. Peto?

A. I can't be sure which one is which, but published studies.

Q. So second question. You were asked whether or not there are distinctions between chrysotile and crocidolite and amosite in terms of potency, and you agreed. As you calculate the risk, or in your case if you presume to calculate the cause to Mr. Cheshire and Mr. Haskins, you still cannot do so with a precise mathematical, arithmetical, numerical exposure level for either of those gentlemen because you don't know it, right?

A. Yes. And because it's not important.

Q. Right. Thank you.

MR. FUSCO: No further questions, Your Honor.

THE COURT: All right.

MR. MERIWETHER: Your Honor?

THE COURT: Yeah.

MR. MERIWETHER: We should make a point to you, as well, that our original motion covered Dr. Bedrossian and covered Dr. Brody. And my understanding was that --

MR. GEORGE: Dr. Brody is not going to offer any specific causation.

MR. MERIWETHER: But in Dr. Brody's report, it very clearly states that each and every exposure causes -- and so any -- any ruling that would come out from this Court on that issue I would ask that we at least address the fact that Dr. Brody's report makes the statement, and it has been withdrawn.

MR. GEORGE: We haven't withdrawn that statement. The fact that each exposure contributes to the cumulative dose is a biological fact. It's not anything that -- it's not an opinion, it's a biological fact that cumulative exposure is made up of all the little baby exposures you had during your lifetime. Now, Dr. Brody is not going to offer any opinion about specific causation. He's there as a general causation witness only, and I'm not going to ask him

about what the effect is of each and every exposure. So I think it's a non-issue. It's not something that he's intending to present.

MR. MERIWETHER: I think it's thoroughly briefed in our motion and their response in Haskins, and I'm not in Cheshire, so I don't know about that. But I was talking about in Haskins.

THE COURT: What do you want?

MR. MERIWETHER: I would -- I would like either for a -- well, I would like either for a ruling to come down that he is prohibited from so testifying because it is scientifically invalid to do so, or a ruling to come down and say they have withdrawn that so he's not going to do it anyway. I just want it clear that we are not going to get to trial in this case and have Dr. Brody talk about each and every or cumulative --

MR. SWETT: It's the same issue, if you recall, we dealt with in the *Sparkman* case. We are not going to ask him about specific causation. The statement they are referring to deals with a medical fact. And the fact is on a medical basis, every exposure contributes to the cumulative dose and the cumulative dose causes disease. We are not going to ask him -- it is not a specific causation issue. That is a medical fact, and it never came up and was never an issue when he came and testified in front of Your Honor a year ago.

THE COURT: So if I understand Mr. Swett, assuming that he was the only expert to testify in the case, that you would get a directed -- the defendants would get a directed verdict because you don't have specific causation.

MR. FINCH: That's correct, Your Honor. If Dr. Brody was all we brought, we would lose.

THE COURT: How is that?

MR. MERIWETHER: Works for me, Your Honor.

THE COURT: Okay.

MR. FUSCO: For us, Your Honor.

MR. MERIWETHER: Feel free to bring just Dr. Brody.

MR. FINCH: I didn't have a trial judge and it was a jury deciding the case, I might do that. But given that there is a man with a robe who gets to decide whether the jury gets to decide the case, I don't think we are going to do that.

Your Honor, just as a matter of housekeeping, we would offer, for purposes of the *Daubert* hearing, the literature.

THE COURT: Wait a second. Dr. Bedrossian, if you want to go down, you are welcome, or you can stay there if you want to.

THE WITNESS: Okay. I'm trying to see if I can carry all of this.

THE COURT: That's fine.

MR. FINCH: We provided Your Honor with copies of the literature which is cited in the PowerPoint. We provided Your Honor a copy of the PowerPoints, you have the reports. For purposes of the *Daubert* record, I would ask that that be included as, whether you want to call it Court Exhibit Number 1, just so you have that material. We don't really need to put exhibit labels on it, unless Your Honor would prefer us to do that.

THE COURT: I'm fine.

MR. MERIWETHER: Your Honor, we should register our objection to the inclusion of additional materials not referenced in the original report at this time.

MR. FUSCO: Join that objection, Your Honor.

THE COURT: Okay. Anything else? We'll get something out sooner or later.

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I certify that the foregoing is a correct transcript from the record of proceedings in the above-titled matter.

Amy C. Diaz, RPR, CRR April 4, 2017

S/ Amy Diaz